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The Clinical Study and
Treatment of Sick Children

THE CLINICAL STUDY AND TREATMENT OF SICK CHILDREN

BY

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M.D., LL.D., F.R.C.P. (LOND. & ED.)

FIFTH EDITION—RE-WRITTEN AND ENLARGED

BY

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PREFACE TO THE FIFTH EDITION

THERE is no greater privilege than to be permitted to help in the perpetuation of the teaching and influence of one's master. This I consider has been my opportunity in the preparation of the present edition of *The Clinical Study and Treatment of Sick Children*, and I need not say that the work has been a labour of love. I also feel grateful for the occasion to further weld the Medical Schools of Edinburgh and Glasgow, and to repay in some small measure the debt that I and the Glasgow School owe to our sister School in the East for some of our most stimulating of teachers.

The first edition of this text-book appeared in 1898, the second in 1908, the third in 1921, and the fourth and last in 1925. These facts are ample evidence that the volume was gaining in popularity and was supplying a very definite want. That it should have met with so much success is not to be wondered at, since it was for the most part the record of the experience of a master clinician who had devoted his life to the study of disease as influenced by, and reflected in infancy and childhood.

Dr John Thomson held very strongly that pædiatrics was not a specialty in the true sense of the term, but that it was merely an extension of general medicine. Thus the volume does not pretend to be a complete treatise of all disease as met with in the child, but is limited to a consideration of special varieties of morbid processes as modified, both in their course and appearance, by the growing patient, and of the methods of physical examination which are applicable in the very young. The book, therefore, is more of the nature of an appendix to works on general medicine and a guide to the student who has already learned the principles of general medicine and acquired the art of physical diagnosis as well as a knowledge of the life history of disease. It is, indeed, this outlook which gives the book its importance.

As it is seven years since the last edition appeared, a certain amount of revision has been required. This was particularly so with regard to nutritional disease in infancy, rickets, the so-called acid-intoxications, the rheumatic infection in childhood, and tuberculosis. It has been my endeavour in the revision to retain as much as possible of the teaching and spirit of the original author and I hope that the emendations and additions have not tended to the production of a disjointed exhibition of the subject.

Naturally, in a work of such a nature, one is indebted for the sympathetic criticism and help of many friends, and I take this opportunity of gratefully acknowledging the assistance given in many directions by my colleagues at the Royal Hospital for Sick Children, Glasgow, and the Princess Elizabeth of York Hospital for Children, London. The radiologists of these respective institutions, Dr D. Campbell Suttie and Dr Bernard Leggett, have through their co-operation made it possible for me to enhance the value of the volume by the inclusion of many radiograms. Professor John Fraser of Edinburgh has kindly revised the section on the cause and treatment of hydrocephalus, which he prepared for the last edition. To Miss Cicely B. Kelly I am indebted for several paintings of radiological appearances and pathological material (Figs. 99, 100, 101, and 102). To Dr M. Witkin of Johannesburg I tender my thanks for drawing attention to errors in the former edition and for suggestions as to how the book could be made more useful.

I acknowledge with thanks the permission of the Controller of H.M. Stationery Office to reproduce several illustrations (Figs. 18, 19, 76, 87, 91, 92, 94, and 95) which originally appeared in Special Report Series of the Medical Research Council; and I am grateful to the Editors of *The Practitioner* for the use of Figs. 66 and 67, to the Editors of *The Archives of Disease in Childhood* for permitting me to again publish Figs. 201, 202, 203, 204, 205, 206 and 207, which in the first instance appeared in their *Journals*, to the Oxford Medical Press for the loan of blocks of illustrations (Figs. 305, 310, 311, 313, 315, 316, 318, 326, 327, 328, and 329) in my book on *Syphilis in Childhood*, to Messrs Edward Arnold for permission to reproduce a Table (p. 897) out of my book on *The Rheumatic Infection in Childhood*, to Dr A. V. Neale for the loan of the block of Fig. 137, to Professor

Alan Brown of Toronto for the photographs from which Fig. 337 was prepared, to Dr Blacklock for permission to publish the Table on p. 952, and to Drs Morris and Macrae for chart for Fig. 97.

To Professor Charles McNeil of Edinburgh and to Dr Hugh B. Thomson, son of the original author, I am under a specially deep debt of gratitude for much valuable advice and assistance in seeing the book through the press.

Finally, I must also express my thanks to the publishers, Messrs Oliver and Boyd, who assisted me in very many ways and who have done everything in their power to make the present edition worthy of its predecessors.

LEONARD FINDLAY.

LONDON, 1933.

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INTRODUCTION

THE subject of the diseases of children is sometimes spoken of as a speciality, but if it is to be regarded as a special field in the domain of medical science at all, it is certainly one which lies beyond, and not parallel to that occupied by ordinary clinical medicine, and it is only through the latter that it can be reached. It is much better not to begin the study of disease in children until familiarity with its symptoms in later life has been acquired.

Infancy and childhood should then be the most interesting periods of life to the physician. It is while they last that his services are most frequently called for, and most indispensable; it is then that he can study disease in its frankest and least complicated forms; and it is then, also, that he is able most satisfactorily to control every circumstance that can act on the patient's health, and therefore ensure the best chance of recovery. It is in little children, too, that the physician's great ally, the *vis medicatrix naturæ*, is present in fullest activity; and consequently, although the mortality is much higher in early childhood than at any other time of life, it is then also that we have by far the largest number of therapeutic successes, and are most frequently cheered by unexpected recoveries.

To the student or practitioner who has hitherto been concerned only with the examination and treatment of adults, the study of disease in childhood is indeed apt to present great difficulties at first, but when these have been surmounted, he finds their ailments at least as easy of diagnosis and treatment as those of his other patients.

To be successful in practice among children, certain qualifications are absolutely necessary to the physician.

Firstly, he must, of course, possess a thorough grasp of the ordinary clinical methods.

Secondly, a certain amount of tact is necessary to enable him to examine the patient without arousing active opposition. Many have this tact instinctively ; some only acquire it in the requisite degree with time and experience ; while to those who have no liking for children, and are out of sympathy with them, it may never come at all.

Lastly, he must have made himself familiar with the chief anatomical and physiological peculiarities of childhood, so as to know what is and what is not within the normal limits ; and he must have some knowledge of the nature and causes of the diseases commonest among children.

In the following pages an attempt is made to supply this necessary information, and to indicate its relation to clinical work, in the hope of arousing some of that interest which, as Dr Moxon said, "neutralises difficulties as alkalies neutralise acids."

THE CLINICAL STUDY AND TREATMENT OF SICK CHILDREN

CHAPTER I

THE GENERAL CLINICAL EXAMINATION, THE CLINICAL HISTORY, AND FACIAL DIAGNOSIS

General Clinical Examination

THE methods of clinical examination in older children are the same as those used for adults and can be applied in the same order. In infants and little children, however, the examination may have to be hastened and its details considerably modified because the patient is unable to endure it for more than a very short time.

In the first place, *our methods differ somewhat in their relative value* in children and adults. Thus, inspection plays a larger part in the diagnosis of disease in infancy than it does in later life. This is partly because it is more easy of application, as children are usually stripped for examination, and are thus more easily seen. Partly also because they show more readily by their gestures and expression what they are feeling and the signs of present disorder are less often in them than in adults obscured by the traces of past disease. Palpation also is much more applicable to children than to grown-up patients, not only because their bodies are smaller and softer, but also because they are so used to being handled all over by their mothers and nurses that it is not to them the unusual and unpleasant process that it is to adults.

There is usually also *considerable difference in the order in which the methods are to be used*. In adults, we generally follow habitually much the same order of procedure—such as is found in the ordinary case-taking forms—although it may be varied somewhat in different cases. In children, the order is changed,

in accordance with the general rule that it is always better to take the more unpleasant parts of the examination last, so that any inevitable crying and resistance may be deferred as long as possible. For example, the mouth and throat are among the last parts to be inspected. Were they examined early, in many cases the child would begin to cry, and the further investigation of his organs be rendered more difficult. Similarly, as some children are frightened by even light percussion, it is better as a rule to auscultate first and to percuss afterwards. Again, it is important to count the pulse and respiration early, before the child has been much disturbed.

As a general rule, while in examining adults we proceed *system by system*, investigating in turn the alimentary, circulatory, respiratory, and other organs, in children we go rather *by methods*, so to speak ; inspecting first as much as we can without touching, then palpating all over, then auscultating, and so on. There are, however, many exceptions to this rule.

Routine Examination.

Before discussing the examination of the different parts of the body, it will be well to describe briefly the investigation of an ordinary case so as to illustrate the usual order of proceeding. Of course most physicians have their own order, and that given here, which is founded on Dr Charles West's chapter on the subject, is only offered as one of many which work well.

To begin with, then, preferably before the child is seen, it is essential to make careful *preliminary inquiries* into his history and symptoms, so as to know to what point especially attention should be directed. It is also advisable that the child should be stripped before the examination is begun. If he is hurriedly undressed in the presence of a stranger it is almost sure to make him cross. It is best, therefore, if possible, to begin with, to have him rolled in a blanket, with or without a loose night-dress on, and on his mother's or nurse's knee. Little children, even when they are seriously ill and feverish, are generally better there than in bed, because they feel safer and are less easily put out. If the child is able to sit up he should always be examined as much as possible sitting, and only subjected to the annoyance of being laid down when it seems quite necessary.

The investigation commences with *inspection*, but it is very

important not to go close to the child and stare at him. If you do, he is very likely to cry. Sit first a little way off and finish your conversation with his mother or nurse. While you are doing so, unobserved by him, notice as much as you can. Observe his demeanour and expression, the state of his development and nutrition, his complexion, the condition of his skin and hair, and the form of his head and any other uncovered parts; also, whether he has signs of rickets or syphilis, or any other disease past or present. Notice whether he is listless or lively, good-natured or irritable, and whether his behaviour is that of a normal child or in any way abnormal. Note also if his respiration is difficult; and, if he cries or coughs, observe the character of the sounds he makes, and count the respirations.

Then comes *palpation*, and by this time the child has got more or less used to your presence, and may allow you to feel his pulse without apparently noticing that you are doing so. Even when a finger on his wrist upsets him, he will probably not object to your counting the pulse-rate by listening through the clothes with a binaural stethoscope. It is essential to count the pulse without annoying or frightening him, because otherwise it will be so quickened by his emotional excitement that its enumeration will be useless.

You will next lay your hand (*thoroughly warm, of course*) on the abdomen, before the child is laid down or his body uncovered. While palpating thus you learn the condition of the skin, its temperature (approximately), and whether it is soft and normal, or dry and harsh as in chronic wasting disease. On deeper palpation you will learn something of the size of the liver and spleen, and the presence of any abnormal swelling or tenderness. The hand may then be passed over the thorax, where you will feel the amount of rickety beading, if any be present, the position and character of the heart's impulse, and if there is any epigastric pulsation. You may also find rhonchal fremitus over the lungs or a thrill over the heart. The limbs should then be felt, and their size, muscularity, and other characteristics estimated.

The hand is then passed over the head, its temperature and degree of moisture noticed, and the state of the fontanelle and the presence or absence of craniotabes investigated. The neck also should be felt, and the exact position of any enlarged lymphatic glands made out.

Auscultation, if the child is nervous (and clean), may be practised first by the ear being laid to the chest with only the nightdress or a handkerchief intervening. For thorough examination, however, the stethoscope should always be used, and a binaural with a short chest-piece (or, preferably, with Bowles' flat chest-piece, which slips easily under the clothes and round the sides) is the most useful form.

It is well to auscultate the back first, as it is the commonest site of many lesions (*e.g.* empyema and collapse). It is also the place where fluid sounds in the bronchi are most likely to be heard, if the infant, as is probable, has been lying down.

Percussion follows auscultation. It must always be light, and care must be taken to see that the child is sitting straight.

The nose and ears should be examined for the presence of discharge; and, last of all, the mouth and tongue inspected, and the gums and fauces seen and, if necessary, felt. This is usually the most unpleasant part of the whole process from the child's point of view, and is therefore best deferred until the end. The temperature may be taken before the examination begins, or at any period during it.

Should the child be *asleep*, it is important to examine him before he wakes, so far as is possible, noting the character of his sleep, whether quiet or restless, and the attitude he assumes (see p. 38). It is also very important to count the respiration and feel the pulse before the child wakes, as this may be the only opportunity for ascertaining their undisturbed condition. If he has any noises accompanying his breathing when awake, note if they are also present during sleep, and if so, to what degree. In many cases the abdomen may be palpated freely and the heart auscultated during sleep; some children are not aroused even by the use of the ophthalmoscope. If the child has to be wakened, this should be done by the mother or nurse, and in any case a strange face should not, if possible, be the first to meet his eye.

It is important to remember that no method of physical examination available in the adult is not practicable in the child. There may be a call for smaller instruments, *e.g.* baby cystoscope and auriscope, and hence a need for greater dexterity. It was only when it was appreciated that a child could be examined like an adult that progress in pædiatrics was made. According to Roger and Charles West, before 1847 auscultation

of children was not practised as a routine, with the result that cardiac disease was not recognised and was looked upon as a rare condition at this age.

Clinical History.

The importance of a full and accurate clinical history can scarcely be overestimated. It makes the physical examination easier and shorter, and saves us from many mistakes. Such a history is generally far more easily obtained in the case of children than in that of adults. Of course, as is so constantly pointed out, the child cannot tell us much about his own troubles. On the other hand, we can usually get our facts from his mother, who has watched him hourly, and to whom his least symptom is a matter of absorbing interest. Her story, if often rambling, is at least more trustworthy than much that adult patients tell us about themselves.

The theories of the mother and nurse as to the cause of the child's illness are usually of no value, but their opinions as to whether he is getting better or worse are never to be made light of. While, however, we may often rely on the mother's description of past symptoms, we must never trust to her account of the urine or fæces, or of any fact of the case which we can investigate for ourselves.

A "method of case-taking" is certainly helpful in clinical work. The Form in use at the Edinburgh Children's Hospital will be found in Appendix A. A few remarks may be made on some of the questions to be asked.

Mother's Complaints.—It is always important, to begin with, to ascertain why the mother has brought the child for advice—which of his ailments, that is to say, bulks most largely in *her* estimation.

Family History.—The family history is also important in some cases, especially in connection with the diagnosis of tuberculous, rheumatic, and nervous cases. We must also inquire about the mother's health, particularly whether she has had any miscarriages, and if she was well during her pregnancy. The number of other children should also be ascertained, and where the patient comes in the family; and it is well to find out whether any of the other children have suffered from tuberculosis, congenital syphilis, or any other special disease.

Previous Health and Treatment.—In investigating the former medical history of a case, you should begin by asking about the nature of the labour and the appearance and behaviour of the child at birth. His growth and development of body and mind must then be inquired into, when his *teeth* appeared, and when he first began to *walk* and to *talk*. It is of great importance also in young children to know about the previous feeding. Whether the child was on the breast, and if so, for how long; if not, what he was given instead of the breast-milk, and at what age he first got solid food. It is also important to know what sort of diet he has recently been getting.

You will then inquire about previous symptoms of disease. A history of “snuffles” and a characteristic rash in early infancy may be important as indicating congenital syphilis. Recurrent attacks of bronchitis, with alternate constipation and diarrhœa and muscular debility, suggest the probability of previous rickets. The occurrence and dates of attacks of infectious disease are always important. In the case of obscure acute symptoms, the fact of the patient’s having been recently exposed to the infection of one of the exanthemata may greatly aid in the diagnosis, and the periods of incubation of these must be kept in mind.¹ Sometimes the fact that an attack of measles or whooping-cough has preceded an obscure illness by a few months is a point in favour of the case being one of tuberculosis.

The Present Illness.—It is often very difficult to get a clear account of the present illness. It is best to begin by ascertaining its duration, and this is done by finding out the exact date when the child was last evidently in his normal state; and then to ask about the ways in which he has since shown that he is not well—his sleep, appetite, energy, temper, appearance, and complaints.

Facial Diagnosis.

Before entering upon a description of the different organs and parts of the body, it will be well to consider briefly the advantage to be gained from a study of the appearance and expression of the child’s face, and the attitude of his body and limbs. Facial diagnosis is easier in children than in older people, because the child’s face is comparatively free from those lines and furrows which are regarded in adults as denoting

¹ See Appendix B.

character; and consequently it is the more easy to read when it bears the impress of disease. It is also specially useful in children, because it frequently tells us, at once, which organ most needs investigation, and so shortens our examination; and because it often affords us the only satisfactory means we have of answering the important question, "How is the child feeling?" The answer to this question may be very helpful. If, for example, the child is obviously feeling well, it is little likely that he is suffering from scurvy, anal fissure, or acute pyelo-nephritis, diseases which usually give rise to much pain.

While facial diagnosis, however, should be practised on all occasions, it is extremely important not to trust to it for such information as can only be acquired with accuracy by the ordinary more laborious methods of examination. If it is employed as a substitute for, instead of as an introduction to, a more methodical examination, the result will be far from satisfactory. Skill in reading the significance of the gestures and facial changes is only acquired by long practice. A few of the changes which are most frequently met with are well seen in the accompanying photographs. In indicating some of the more important points to be observed free use is made of Professor Soltmann's valuable paper on the subject.¹

Pleuro-pneumonia.—The first photograph (Fig. 1) is of a boy of 22 months taken on the fifth day of an attack of acute pleuro-pneumonia which involved part of the base of the right lung.

The child is too ill to notice much or to hold up his head, which is lying back on his mother's arm. His face is flushed and his eyes bright, although their expression is dull and anxious. His eyebrows are oblique from the action of the corrugator supercilii on each side along with that of the central bundle of the frontalis. His nostrils are dilated and working, the angles of the mouth are lowered and the lips slightly parted so as to admit more air during the laboured breathing. The general expression of the child's face is that of suffering, modified by the desire not to cry because of the pain which a long breath would cause.

Fig. 2 represents the same child nine days later, after the

¹ "Ueber das Mienen- und Geberdenspiel kranker Kinder," *Jahrbuch für Kinderheilkunde*, 1887, xxvi., 206.

temperature had been normal for more than a week. The abnormal points in Fig. 1 become much more evident when you compare it with this one. Here the child is thinner, but his general expression is that of health, comfort, and intelligent observation.



FIG. 1.—Pleuro-pneumonia, before the crisis. (Boy of 22 months)



FIG. 2.—Pleuro-pneumonia. The same child a week after the crisis.

Meningitis.—Figs. 3 to 7 illustrate the physiognomy of brain disease. This is an important facies to recognise, because in so many cases the other symptoms of brain disease are equivocal during the early stage, and it may be doubtful whether the lesion is in the head, the chest, or the abdomen. Under these circumstances much may be learned from the expression of the face.

The look that brain disease gives to a baby's face is a very strange one, because it suggests the presence of emotions which are quite foreign to infancy. This is seen in a striking way in Figs. 3 and 5, which represent a baby of three months who was dying of basic meningitis.

His eyes are closed, and he is knitting his brows. According

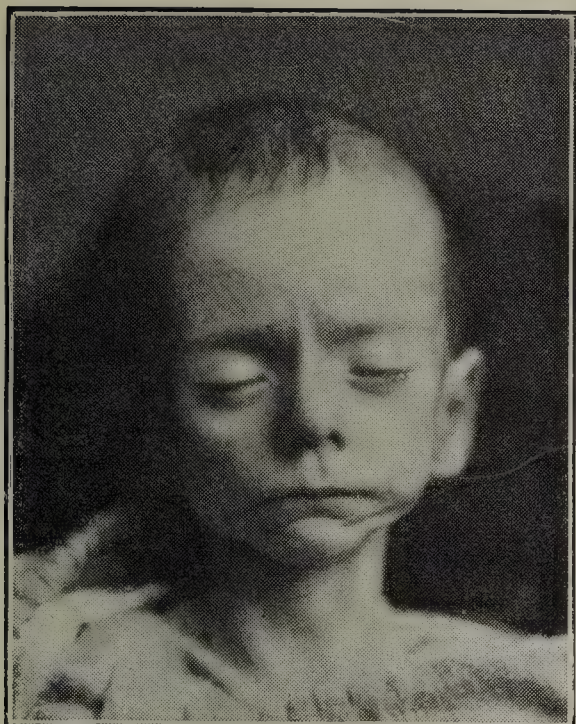


FIG. 3.—Moribund Basic Meningitis.
(Boy of 3 months.)

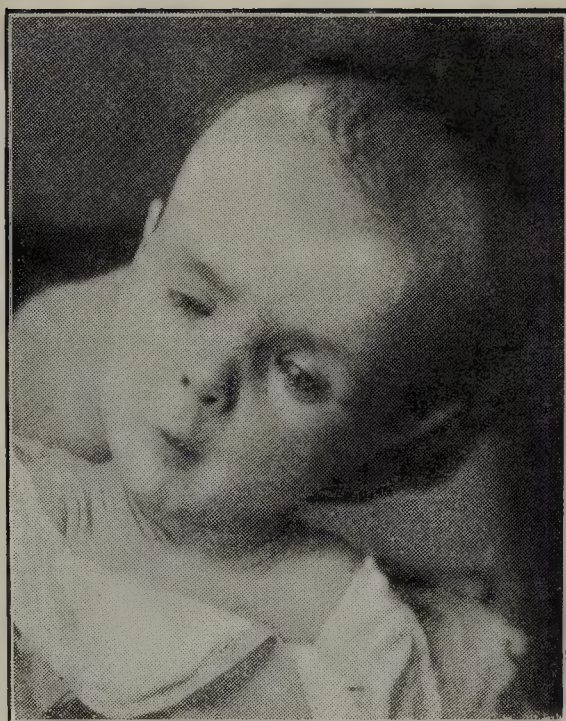


FIG. 4.—Tuberculous Meningitis.
(Girl of 11 months.)

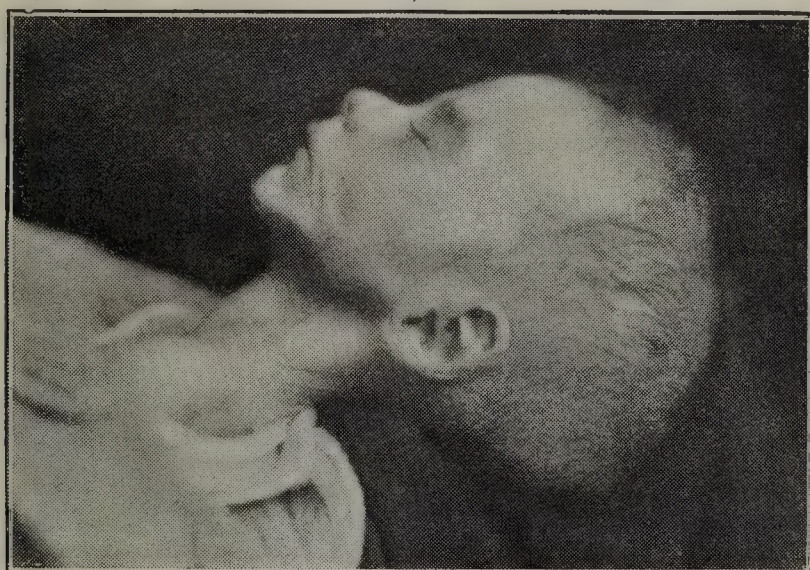
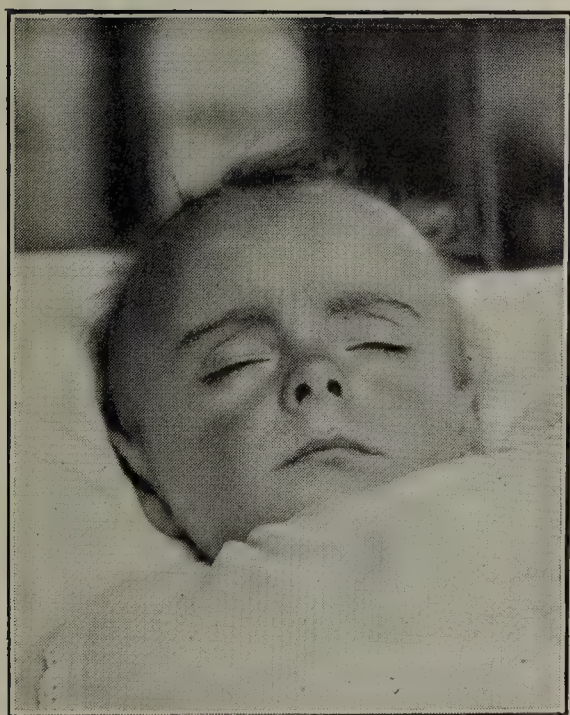


FIG. 5.—Same as Fig. 3.



FIGS. 6 and 7.—Tuberculous Meningitis. (Boy of 2 years.)

to Professor Soltmann, it is the hyperæmia of the retina associated with the brain condition which produces this effect on the face, just as a bright light might do. The central irritation is producing very tight contraction of the masseters, and there is also some retraction of the head. The general aspect of the upper half of the face is that of deep and earnest thought, while the clenched jaw and slight drawing back of the head give almost the impression of stern determination. Although the baby is much emaciated his fontanelle is full and bulging.

Figs. 4, 6, and 7 represent infants suffering from tuberculous meningitis. The unchildlike look of profound meditation, as well as (in Fig. 4) the squint and unequal opening of the sightless eyes, are very noticeable.



FIG. 8.—Acute Suppurative Peritonitis.
(Girl of 19 months.)

Facies of Acute General Peritonitis. — In cases of abdominal suppuration (appendicitis, etc.) where the infection has spread to the general peritoneum, a facies is often seen which is apt to be mistaken for that of meningitis. A good instance

of this is represented in Fig. 8. Note the sunken eyes and the drawing down of the angles of the mouth. When this facies is well marked it generally indicates that the time for successful surgical interference has passed.

Risus Sardonius.—This is seen in children with tetanus, and is helpful in the diagnosis in the early stages of slight cases (Figs. 9 and 10).

The Hippocratic Facies of Impending Death.—Fig. 11 represents an infant, aged 10 months, dying from severe septicæmia with diarrhœa and broncho-pneumonia. The child was evidently delirious, grasping in the air in front of her, as if she saw things. Note the hollow staring eyes, the dilated nostrils, and the dropping of the lower jaw. This photograph may be taken as representing the “*facies Hippocratica*” as it is seen in an infant. Hippocrates, in describing a dying man, speaks of the “sharp nose, hollow eyes, collapsed temples, the ears cold, contracted, and their lobes turned out. The skin

about the forehead being rough, distended, and parched; the colour of the whole face being green, black, livid, or lead-coloured."



FIG. 9.—Tetanus. (Boy of 5 years.)



FIG. 10.—Tetanus. (Girl of 3½ years.)

Adenoid Facies.—Fig. 12 represents the common appearance of the face seen in children suffering from obstruction of the naso-pharynx by adenoid vegetations. There is a general dull expression, and the mouth is almost constantly open. The nostrils are very narrow, the alæ nasi are defective



FIG. 11.—Facies Hippocratica.
(Girl of 10 months.)

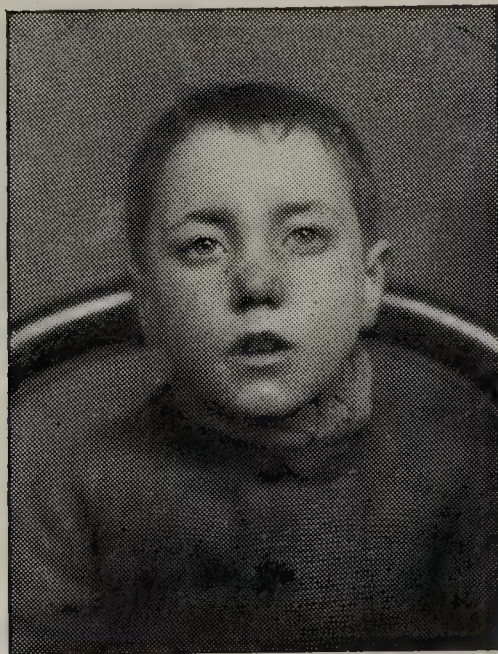


FIG. 12.—Adenoid Facies.

and present a more or less distinct dimple at the junction of the superior and inferior lateral cartilages. Owing to accompanying nasal catarrh there is often, as there was in this case, a tendency to running at the nose, and its extremity is apt to be red, especially in cold weather.

The degree to which the adenoid physiognomy is present indicates the amount of obstruction to the process of nasal breathing rather than the amount of the adenoid vegetations. The latter may be present in considerable bulk, and may be doing harm in reflex and other ways; but if they do not obstruct the free entrance of air through the naso-pharynx they will not cause this alteration of the facial appearance.

Asthma, etc.—In some cases of asthma and in cases of severe chest deformity (*e.g.* from Pott's disease), in which the



FIG. 13.—Acute Nephritis.



FIG. 14.—Same child after recovery.

extraordinary muscles of respiration are habitually over-acting, there is an opposite condition to the adenoid physiognomy. In children with such affections the eyes tend to be unusually bright, the nostrils large, well developed, and widely open, and the mouth firmly closed.

Acute Bright's Disease.—Fig. 13 shows the swollen and puffy appearance of the face characteristic of acute nephritis, and Fig. 14 the same child after recovery.

Acute Diarrhoea and Vomiting.—Fig. 15 shows the staring eyes, with deep hollows round them, which are characteristic of babies with acute vomiting and diarrhoea. This facies is the

exact opposite of that in acute nephritis; for here the tissues of the face and orbit, instead of being water-logged as in nephritis, are shrunken for want of the fluids of which the acute vomiting and diarrhœa have deprived them. In cases such as this the subcutaneous injection of saline solution will very rapidly bring a more normal appearance to the face, besides raising the level of the sunken fontanelle.

Chronic Diarrhœa. — Children with chronic diarrhœa often have an expression of disgust and aversion. It is probable that this is merely the result of a constant bad taste in the mouth in a child rendered weak and irritable by exhausting disease.

Scurvy. — This disease is characterised by extreme pain on movement of the limbs; hence the child has an expression of terror when anyone approaches, because of the fear that he is going to be moved. Hæmorrhage into the orbit is also characteristic of scurvy (Fig. 75, p. 236).

Pyelo-nephritis. — The infant suffering from pyelo-nephritis also has often a characteristic facies. An extreme degree of pallor and an expression of obvious discomfort are the predominant features.

Malignant Medullary Hypernephromata. — Owing to the fact that metastases in the flat bones are liable to occur, irregularities of the skull, proptosis with hæmorrhage into the orbit, and facial paralysis are not uncommon and produce a not uncharacteristic appearance (Figs. 168 and 169, p. 522).

Chloroma. — In this variety of leukæmia tumours of the cranial bones are also present, and a picture similar to that met with in malignant medullary hypernephroma is produced (Figs. 144 and 145, p. 485).

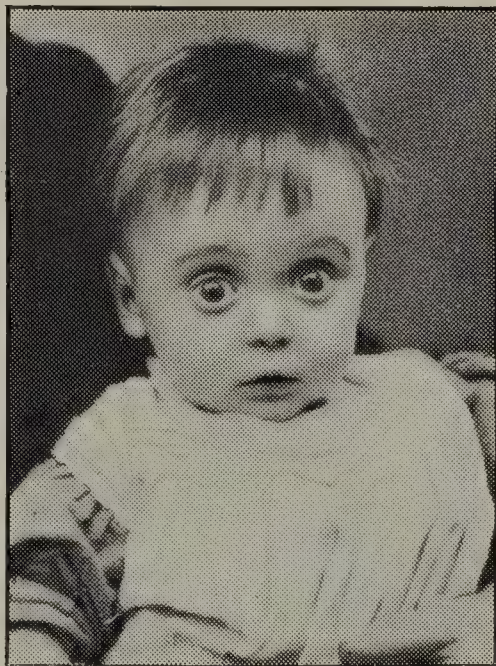


FIG. 15.—Acute Diarrhœa and Vomiting. (Girl of 11 months.)

CHAPTER II

GROWTH AND DEVELOPMENT

Growth in Weight

THE average weight of a new-born baby is 7 lb. or thereby (5 to 12). During the first two days of life there is usually a loss which may amount to as much as 8 or 10 oz. This is partly due to the passage of urine and meconium, but mainly to the fact that the child does not receive enough nourishment from the breasts to make up for the tissue waste. If the child is satisfactorily fed from the beginning there may be no loss of weight. On the third day a steady rise usually begins and the birth-weight is reached again, on an average, by the tenth day of life.

After this the rise continues more or less steadily, the infant gaining from $\frac{2}{3}$ to 1 oz. daily during the first five months, and from $\frac{1}{3}$ to $\frac{2}{3}$ oz. daily during the rest of the first year. The gain may vary from day to day, but the average daily increase for the week will be at about that rate.

By the end of the sixth month the baby's weight should be a little more than double what it was at birth, and by the end of the first year about three times the original figure. During the second year the child gains 5 to 6 lb.; during the third, fourth, fifth, sixth, and seventh years about $4\frac{1}{2}$ lb. a year; and during the eighth, ninth, tenth, and eleventh years 6 lb. per year. By the end of the sixth year the weight is somewhere about six times the birth-weight, and by fourteen years double that figure. The average weight at any age may be seen from the accompanying charts (Figs. 16, 17, 22, and 23).

An infant who is very small at birth generally takes a long time to reach the average weight; and a bottle-fed child gains more slowly, other things being equal, than a breast-baby. This latter point is demonstrated in the charts (Figs. 18 and 19) which refer to infants of the working classes in Glasgow and

Edinburgh, studied by Noël Paton and Findlay in their investigation of the relationship between poverty, nutrition, and growth.¹ There are, of course, wide variations within the limits of health.

Clinical Significance.—The advantage, from a clinical point of view, of weighing children regularly is very great. In scientific infant-feeding, and in the treatment of food-disorders,

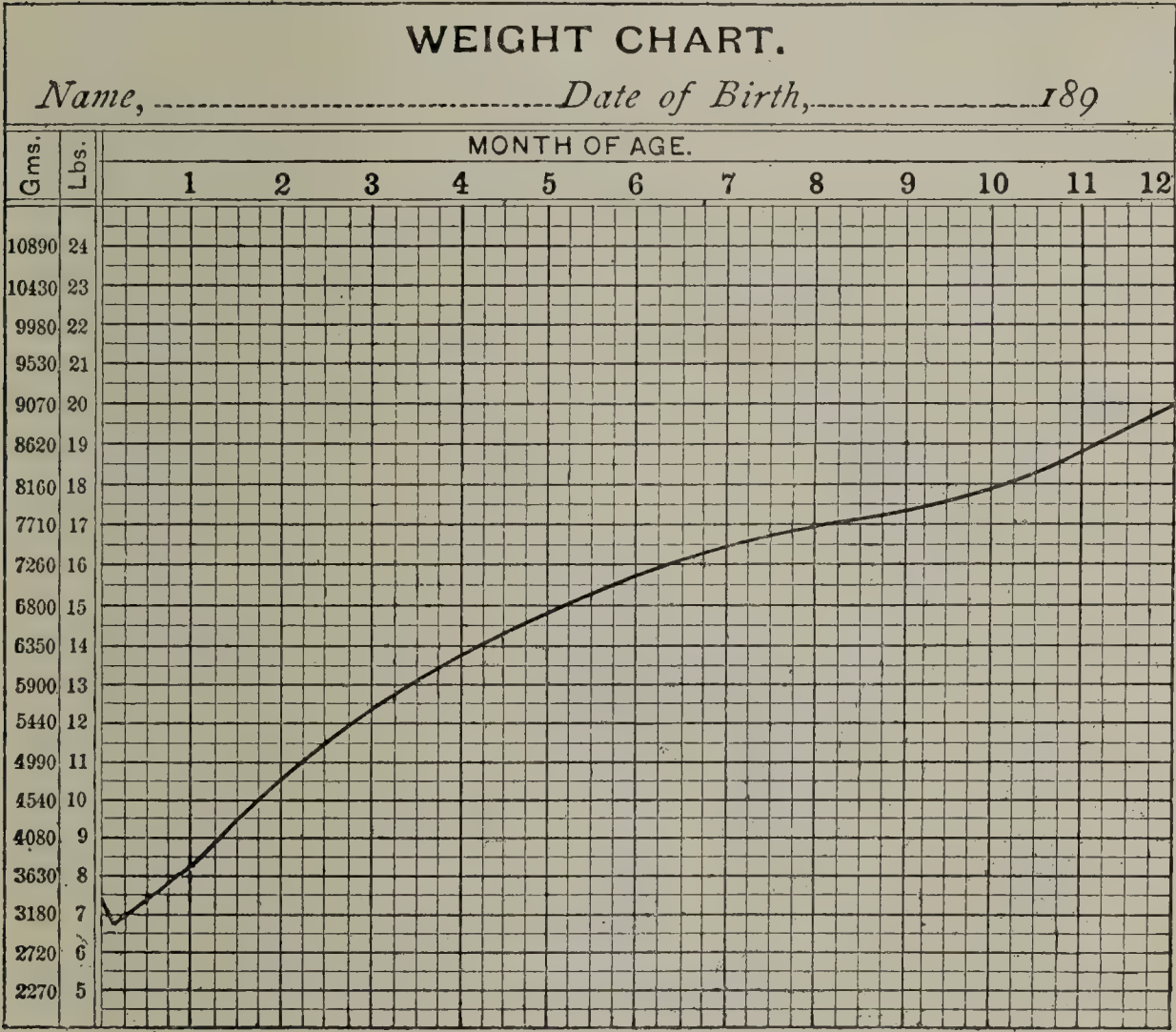


FIG. 16.—The weight curve of the first year (Holt).

it is indeed quite indispensable. The weight should be noted once a week in ordinary cases, and in food-disorders it is often well to take it daily. By observing it we are able to estimate more easily and surely than in any other way the extent to which a particular diet is being assimilated. An interruption in the gain will often show that a new diet is not being properly digested, although it has not, as yet, caused any of the more obvious symptoms of dyspepsia. The course of the weight curve is, in many cases, a source of encouragement; for, if it is

¹ D. Noël Paton and Leonard Findlay, "Poverty, Nutrition, and Growth," *Med. Res. Council Rep.*, No. 101, 1926, 202.

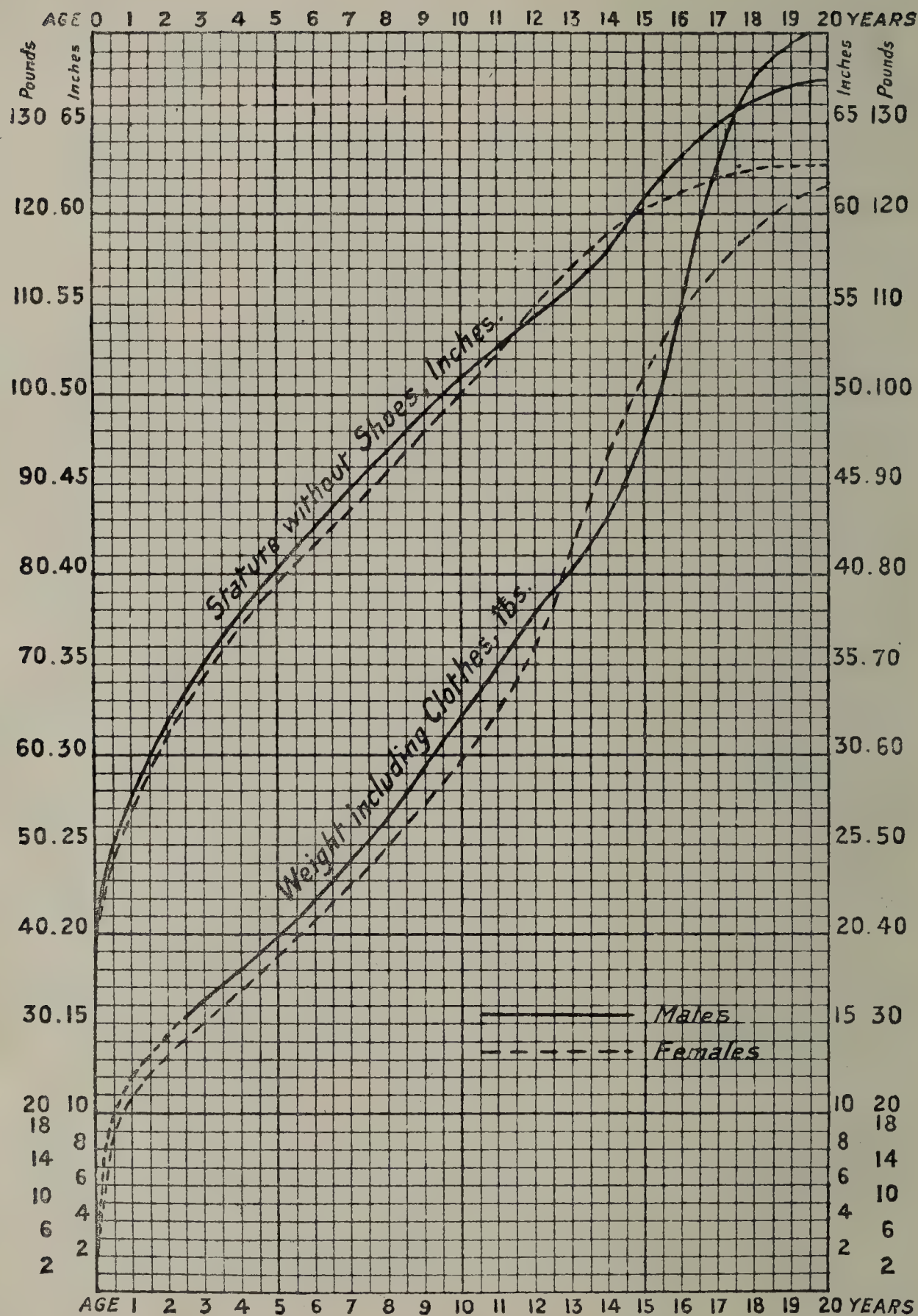


FIG. 17.—Chart showing average stature and weight of both sexes under 20 years.

(From the Report of the Royal Commission on Physical Training (Scotland), vol. i., 1903.)

quite satisfactory in character and the baby vigorous, we may be sure that all is going well with him in spite of any other symptoms that may have aroused anxiety. It is also often an invaluable guide in his treatment.

Various temporary disturbances of mind as well as of body may cause an arrest, for a time, of a child's gain in weight. Some babies, for example, lose during their first week in an hospital ward, without any change of diet; and, afterwards,

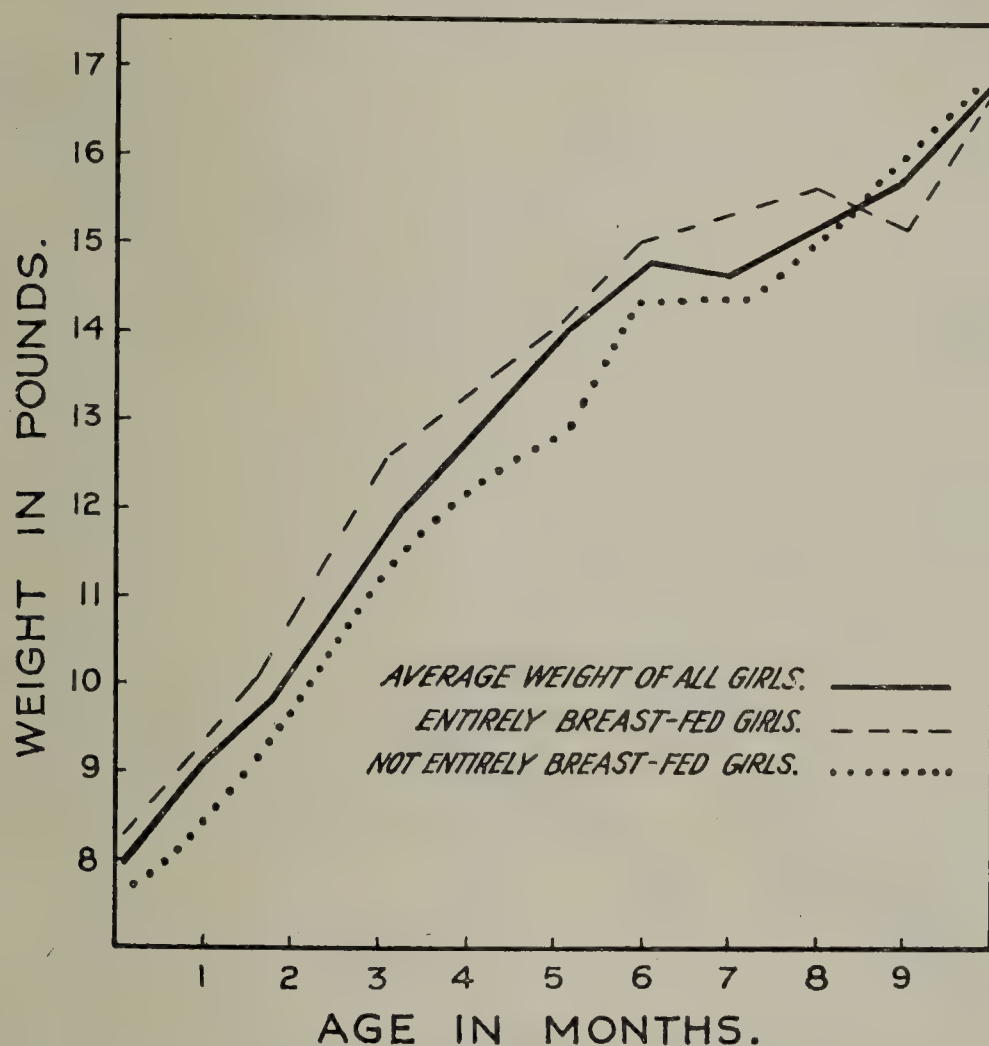


FIG. 18.—Chart showing average weights of girls at different ages, from one week to ten months: (1) entirely breast-fed, and (2) not entirely breast-fed. (From *Poverty, Nutrition, and Growth*, by D. Noël Paton and Leonard Findlay.)

when used to their surroundings, gain satisfactorily on the same treatment. On the other hand, the reverse may happen; the child gains well during the first week or ten days and then, probably on account of the monotony of the hospital regime, ceases to put on weight. Slight ailments of any kind, and also the cutting of teeth, often stop the gain temporarily.

Whenever a young child is losing weight, or even not gaining it for many weeks, this is to be regarded as an important fact, the cause of which must be investigated. It is equivalent to progressive emaciation in the adult, and is

never to be quietly acquiesced in, however free the child seems from other symptoms. A baby that is not gaining weight properly has a lowered resistance to disease, and is failing to lay in the stock of strength by which he is to hold his own in the trials of later life. Such a child, though often regarded as satisfactory by his parents, is really in constant danger

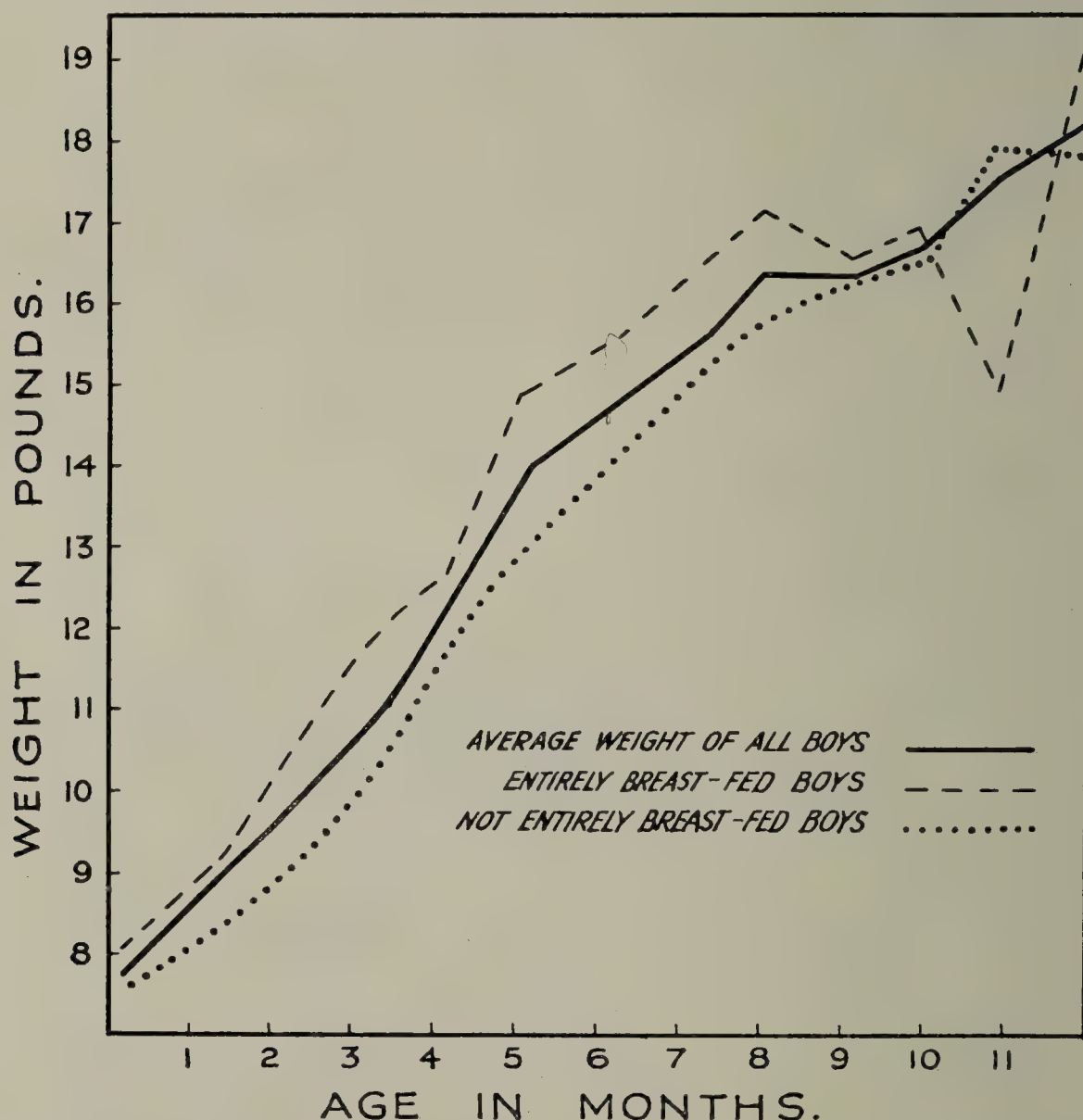


FIG. 19.—Chart showing average weights of boys at different ages, from one week to one year : (1) entirely breast-fed, and (2) not entirely breast-fed. (From *Poverty, Nutrition, and Growth*, by D. Noël Paton and Leonard Findlay.)

of succumbing to some comparatively slight chest or bowel attack which would not have harmed a better nourished baby.

Although, in most cases, a growing weight means that the infant is thriving, we not very rarely meet with instances in which it has a less favourable explanation. Certain forms of feeding cause an abnormal rise in the weight curve. For example, an increase of cane-sugar in the food may do so, the rise being so rapid and considerable that it is obviously

out of proportion to any possible effect which the new food could have had, in the time, on the child's nutrition. This misleading gain is due to the large proportion of sugar in the food having produced in the tissues an increased deposition of glycogen, which combines with a large amount of water. A rise in weight of this sort is often seen in infants who have been gaining slowly on modified cow's milk, when they are given ordinary sweetened condensed milk or one of the sugary proprietary foods. The same result follows an increased intake of salts as when the feed is changed from breast-milk to cow's milk, or from one of diluted to one of undiluted cow's milk. The increase of weight is not usually accompanied by any obvious signs of œdema. A diminution of the sugar in the food or a change from a salt-rich to a salt-poor diet may, similarly, cause a sudden disproportionate drop in the weight curve.

Another common cause of such a retention of fluid in the tissues is an excess of salts in the food. This is often seen when the child is fed on cow's milk. Finkelstein has drawn attention to the striking difference between the steady uninterrupted rise of weight which occurs in normal babies on breast-milk and the zigzag weight curves which are so often seen in artificially-fed infants. He attributes the irregularities in the weight curves of the bottle-fed babies to the alternate retention and excretion of the larger amount of salts in the cow's milk. He has also shown that valuable aid may be obtained from the weight chart in recognising different types of food disorder.

In any weakly baby of a few months who is in an advanced state of marasmus, there is a strong tendency to the occurrence of superficial œdema. When such an infant has shown little or no gain for some time, and then becomes suddenly heavier, we must always look out for puffiness of the skin on the back, face, or extremities. This is a not infrequent occurrence in the late stages of pyloric stenosis.

In infants who are suffering severely from tuberculous disease a sudden gain is to be regarded as suspicious, and should lead us to examine for accumulation of fluid in the subcutaneous tissues or in the peritoneum or pleuræ.

In older children, as well as in infants, a temporary loss of weight is an important sign of many morbid conditions. In institutions in which the children are weighed regularly, as

a matter of routine, it is found that the steady rise of their weight is often checked at once by a severe catarrh or a sore throat. The same thing is noticed during the latter half of the incubation period of measles. Another example of the practical importance of weighing children regularly is afforded in the course of some cases of empyema. Cases of this kind in which there is a small localised collection of pus are not uncommon in childhood. They may, not infrequently, be cured by one or more aspirations. After the pus has been removed, however, it is often impossible to make sure from a physical examination alone whether it is, or is not, reaccumulating. The question can usually be settled by weighing the child periodically. So long as the pus is gathering again the child does not gain weight; and, conversely, if there is a satisfactory gain in weight, without a great increase in the local dullness, we may be sure that there is no need of further tapping.

Emaciation.—Chronic emaciation in young children is generally due to dyspepsia or to improper feeding, but it may be caused by tuberculosis or some other serious chronic disease. In older children it may be the result of tuberculous disease, of diabetes, or of hysteria, or some other nervous affection. Far the commonest cause of persistent failure to gain at this age, however, is chronic intestinal indigestion (p. 298).

Obesity.—When children show an excess of subcutaneous fat it is generally distributed more equally over the body and is less markedly abdominal in situation than is usual in adults. In many cases the excess is due to over-feeding—especially with carbohydrates and fats; and the resulting interference with exercise tends constantly to increase it. There is reason to believe, however, that in many cases and in certain families the obesity is due to a constitutional defect in metabolism, and not merely to too much food. Such defects in metabolism may depend on a morbid condition of one of the endocrine organs, but this should not be concluded unless there is definite evidence of such dysfunction. In many examples by the time the child reaches adolescence he has a normal appearance.

The treatment of simple obesity in children consists in cautious restriction of the diet and in carefully regulated exercise. Over-eating should, of course, be prevented, and the amount of fat in the diet lessened. Sugar should also

be diminished and no sweets given. A reasonable amount of other carbohydrates is advisable and plenty of meat and eggs. The child should have plenty of open-air exercise—short of fatigue. Thyroid gland is rarely indicated except in cases in which there is reason to suspect thyroid deficiency; and no other drugs are of any practical value.

Progressive Lipodystrophia.—In connection with emaciation and obesity a rare and interesting disease may be mentioned, to which the name of “progressive lipodystrophia” has been given,¹ as in many of the published cases the symptoms have begun in childhood. Its chief characteristic consists in a progressive symmetrical loss of subcutaneous fat in the upper half of the body, which is usually most strikingly seen in the face and chest. While these parts of the body waste, the lower limbs show an increasing plumpness, so that the contrast becomes very noticeable. Most of the patients hitherto described have been girls. The muscles remain normal, there is no tendency to infantilism, and no other symptom. The general health is quite unaffected.

Growth in Length.

At birth the child measures, on an average, $19\frac{1}{2}$ to $20\frac{1}{2}$ inches in length. During the first year he grows about 9 inches; during the second, third, fourth, and fifth years on the average 3 inches per year, and during each year from six to twelve years 2 inches. Between the eleventh and fourteenth years girls are rather taller than boys of the same age. By the end of the fifth year the child has generally doubled his original length.

The growth in height shows distinct periodical variations. It is less in boys between nine and fourteen, and in girls between eight and eleven, than it is immediately before or after these ages. It also varies at different seasons of the year, being greatest in the spring and summer months (April to August), less between August and November, and least during the winter.

The average height, at a given age, of children depends on the social class to which they belong, and whether they are

¹ Feer, *Jahrb. f. Kinderheilk.*, 1915, lxxxii., 1; C. Herrman, *Arch. Int. Med.*, April 1916, xvii., 516; F. Parkes Weber, *Quart. Journ. Med.*, 1917, x., No. 38, 131.

reared in the city or in the country. The same is true of the average weight, but not to the same extent. This is well demonstrated in the following charts (Figs. 20 to 23) showing the average heights and weights of children of the well-to-do seen in private practice by one of us (L. F.), of children attending the Board Schools in the city of Glasgow and the Council Schools in the rural districts of Ayrshire. These averages are contrasted with those published by Holt,¹ which refer to children attending public and private schools in the United States, and which are commonly taken as the standard. The better development of the well-to-do class child is clearly shown in these charts, and it is possible that it would have been still better if the children studied had been taken from the school-room rather than from the consulting-room. They also show us that unless different standards are employed for the different social classes erroneous conclusions will be drawn.

Clinical Significance.—The infant's growth in length has not been nearly so fully studied as his gain in weight. Variot² has made special investigations on the subject, and has pointed out various facts regarding it which are of great clinical interest and importance.

The normal baby adds to his length continuously from birth. Although, under ordinary conditions, the increase in weight and that in length usually run a somewhat parallel course, they are really quite independent of one another. This is seen even in the first days of life for, during the rapid loss of weight which usually takes place at that time, the gain in length proceeds very actively, so that he grows $\frac{2}{3}$ inch in the first fortnight and about $1\frac{1}{2}$ inches in the first month; and half of his first year's growth has often occurred by the time he is three months old. In later childhood also there is evidence, from time to time, of a physiological dissociation of the weight and length curves; for the length often continues growing while the weight is stationary, and generally grows most rapidly when the gain in weight is slow.

Although the weight chart gives us all the information we

¹ L. E. Holt, *Diseases of Infancy and Childhood*, New York, 8th edition, 1922, 66.

² *La Clinique Infantile*, 1905, iii., 323; 1908, vi., 385, 496, and 609; 1909, vii., 193; 1911, ix., 217; 1914, xii., 289; also Hubert, *ibid.*, 1910, viii., 462.

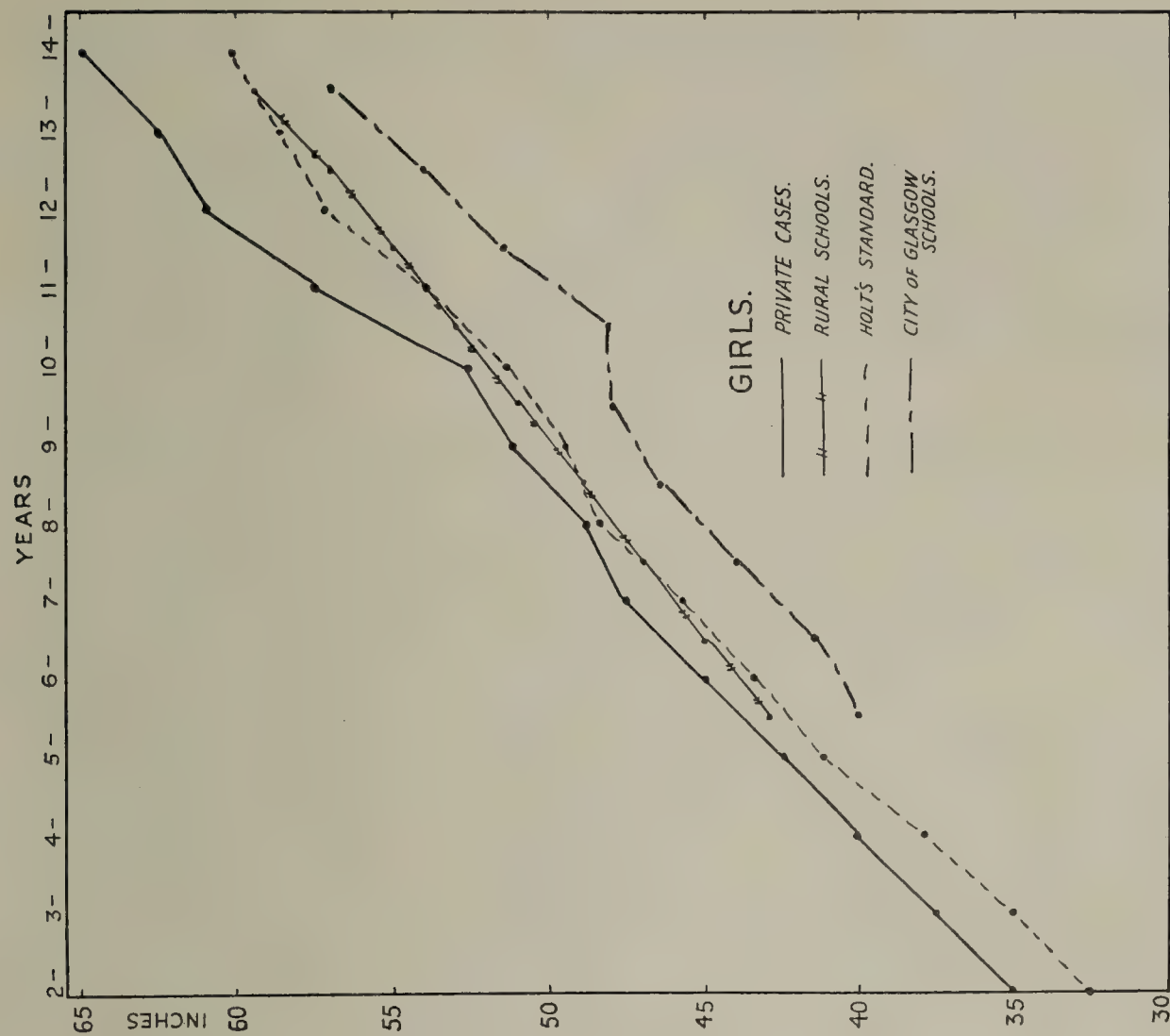


FIG. 21.—Average heights of girls belonging to different social classes.

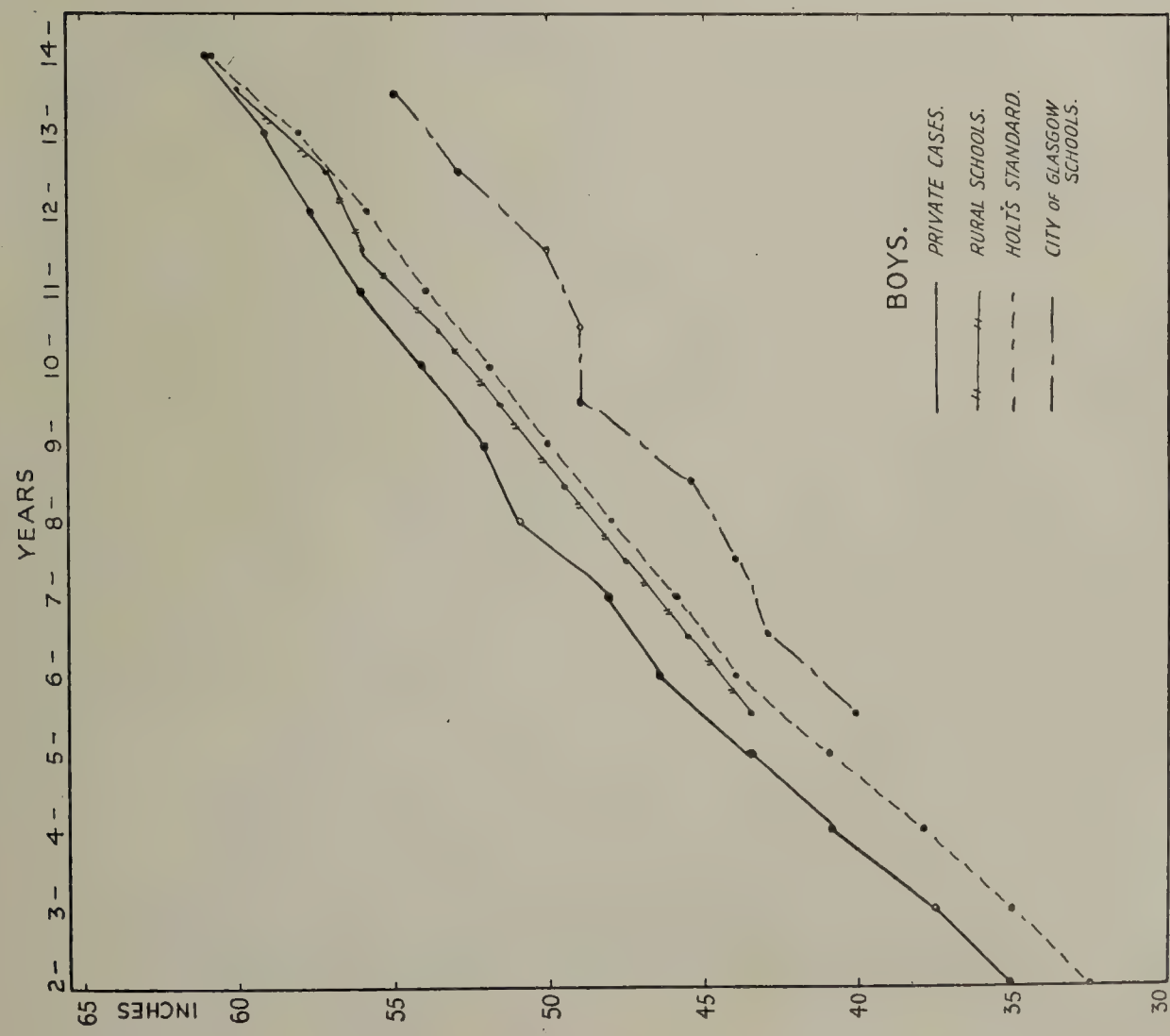


FIG. 20.—Average heights of boys belonging to different social classes.

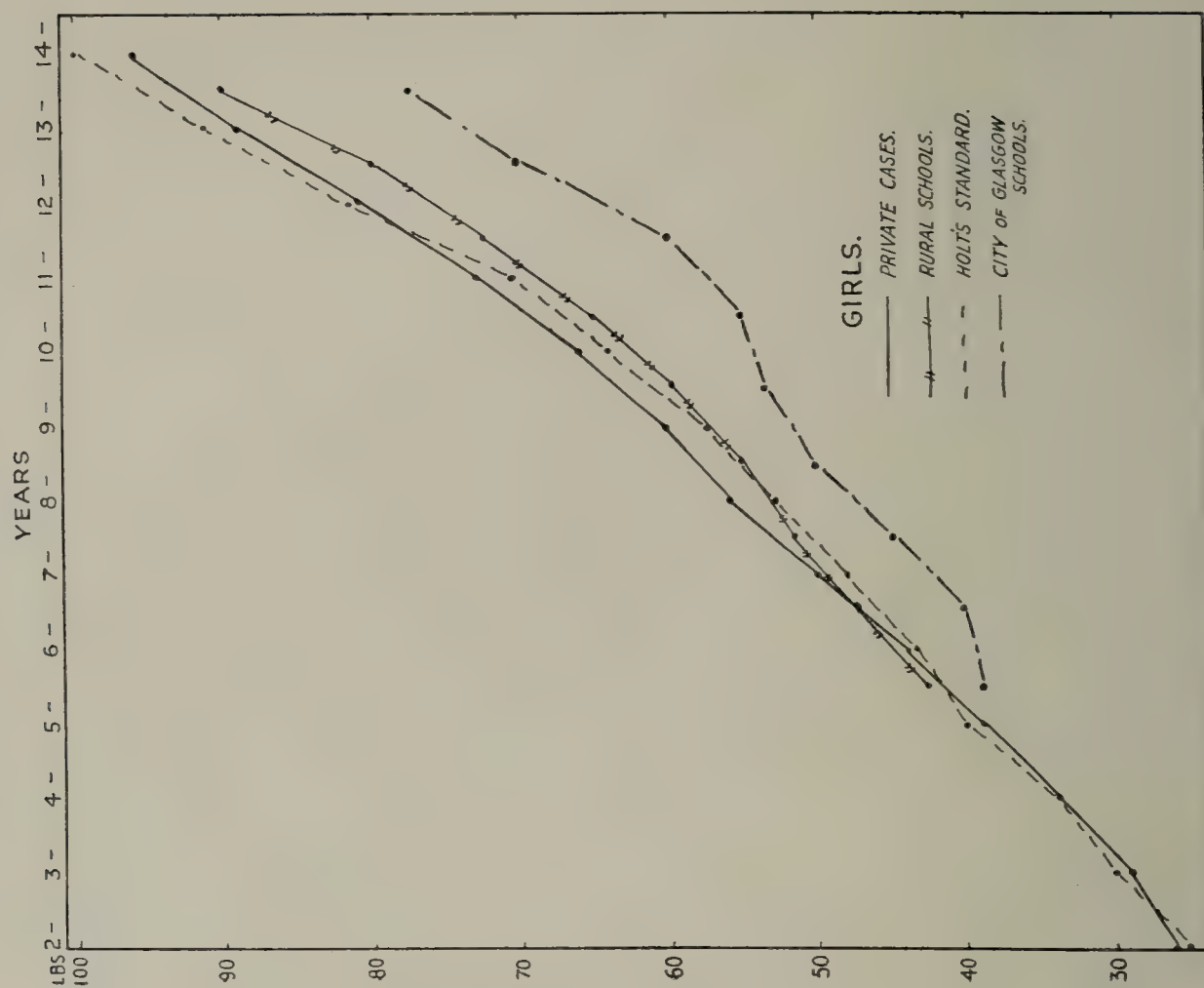


FIG. 23.—Average weights of girls belonging to different social classes.

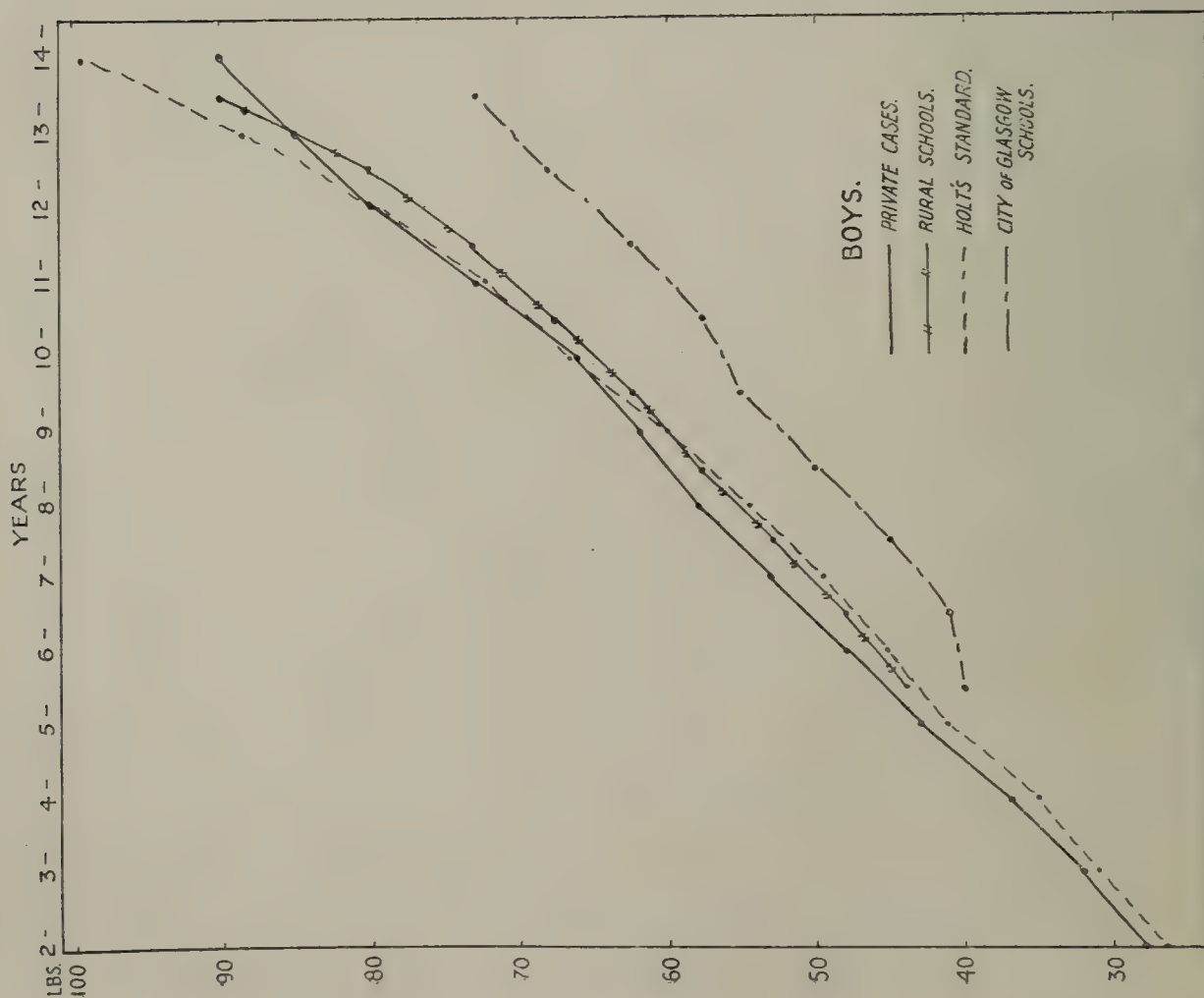


FIG. 22.—Average weights of boys belonging to different social classes.

usually need in estimating the development of the normal baby, it is important in the abnormal infant to take the growth in length into account also. It is in premature and debilitated babies that the dissociation of the growth in weight and length shows most and lasts longest. When a baby is suffering seriously from atrophy, his length increases while his weight falls, just as happens in typhoid and other serious illnesses in later life.

Variot¹ and Lassablière have found that, after the second month, the relation between the growth in length and the nutritive requirements is so constant that they regard the former as a trustworthy guide to the amount of food the child should have. They estimate that 14 grammes of breast-milk are needed for each centimetre of the infant's length.

Although arrest of the growth in length is most often due to gastro-intestinal disturbance, it is also characteristic of various other diseases such as tuberculosis, congenital syphilis, and rickets²; and Hess³ has found that prolonged infantile scurvy also exerts a striking influence of this sort, and that antiscorbutic treatment is followed at once by greatly increased growth in length. Variot⁴ has also pointed out that the date of appearance of the centres of ossification in the bones of the phalanges and metacarpus varies with the growth in length and not with the gain in weight. This is illustrated in a striking way in cretinism and achondroplasia, and in most of the other forms of disease which cause dwarfing, for in them the ossification of these bones is very greatly retarded.

In estimating the state of nutrition in school children, the innumerable observations which have been made regarding the relation of their height and weight to their age have proved of very little practical value. As Holt⁵ has shown, the only really important point to ascertain is the relation of weight to height. This, along with the annual rate of increase in weight and height, and the general appearance and bodily conformation of the child are the chief data from which the examiner can form a just estimate of the nutrition. It must be

¹ *La Clinique Infantile*, 1908, vi., 641 ; 1909, vii., 225.

² George Rose, *Lancet*, Nov. 1924, 1140.

³ *Amer. Journ. Dis. Child.*, Aug. 1916, xii., 165.

⁴ *La Clinique Infantile*, 1906, iv., 685.

⁵ *Amer. Journ. Dis. Child.*, Dec. 1918, xvi., 359.

appreciated, however, that the question of nutrition is quite a different matter to that of development.

In dealing with deficient stature we must, especially in older children, distinguish between simple arrest of growth, or **dwarfing**, and the same condition associated with infantilism. In simple dwarfing the development of the sexual organs, with its accompanying changes in other parts of the body, proceeds

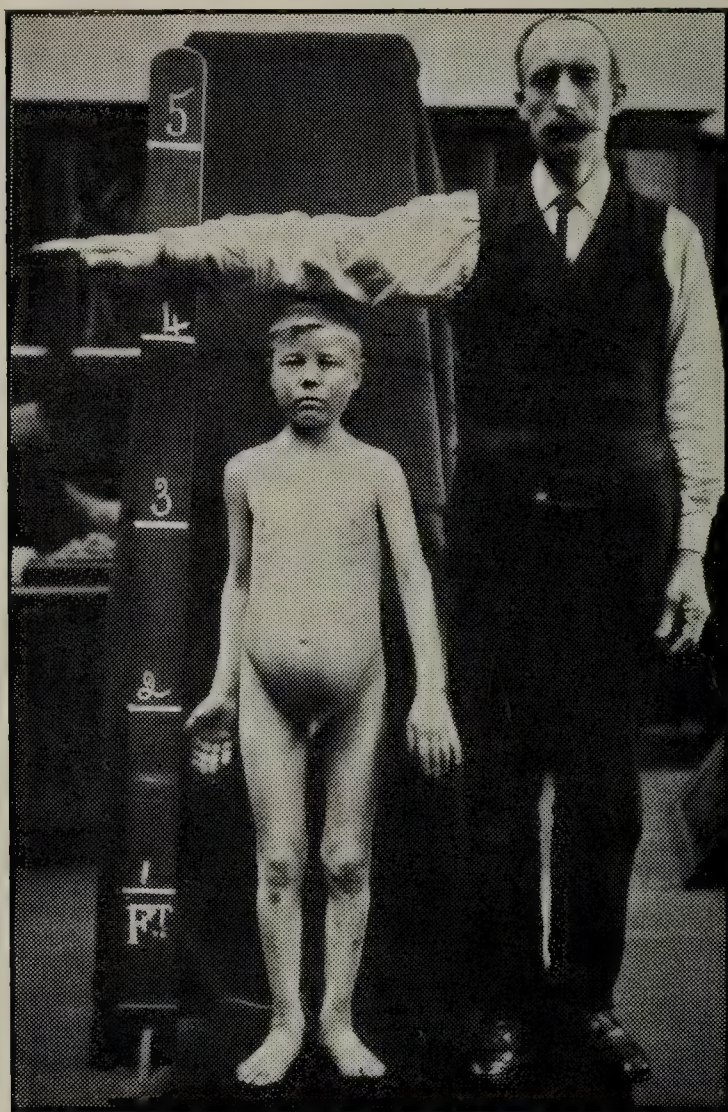


FIG. 24.—Pancreatic Infantilism. (Boy of 25 years.)
Died of diabetes in 33rd year.

in the ordinary way; while in **infantilism** the child retains his infantile proportions and contour; and, as he grows older, there is a delay in those changes in the genital organs, larynx, and elsewhere which announce the approach of puberty. It is generally, though not always, accompanied by dwarfing.

Infantilism is found in an extreme degree in cretinism, and it is one of the symptoms to be looked for in the slight cases of that disease which are so apt to be overlooked. It is also, though to a less extent, characteristic of defect of pituitary secretion. It is not a characteristic of mongolism. Serious

affections of other organs such as the liver,¹ pancreas² (Fig. 24), and kidneys³ may also produce infantilism, and it is one of the most striking manifestations of the form of chronic diarrhoea which is known as "coeliac disease" (p. 303). Delayed sexual development is also found in many other debilitating conditions, such as cardiac malformations, congenital syphilis, and chronic tuberculous bone affections, likewise in some cases of cerebral diplegia, and in some other organic lesions of the brain.

In cases of dwarfing with infantilism for which there is no obvious cause, it is well to try the effect of thyroid in small

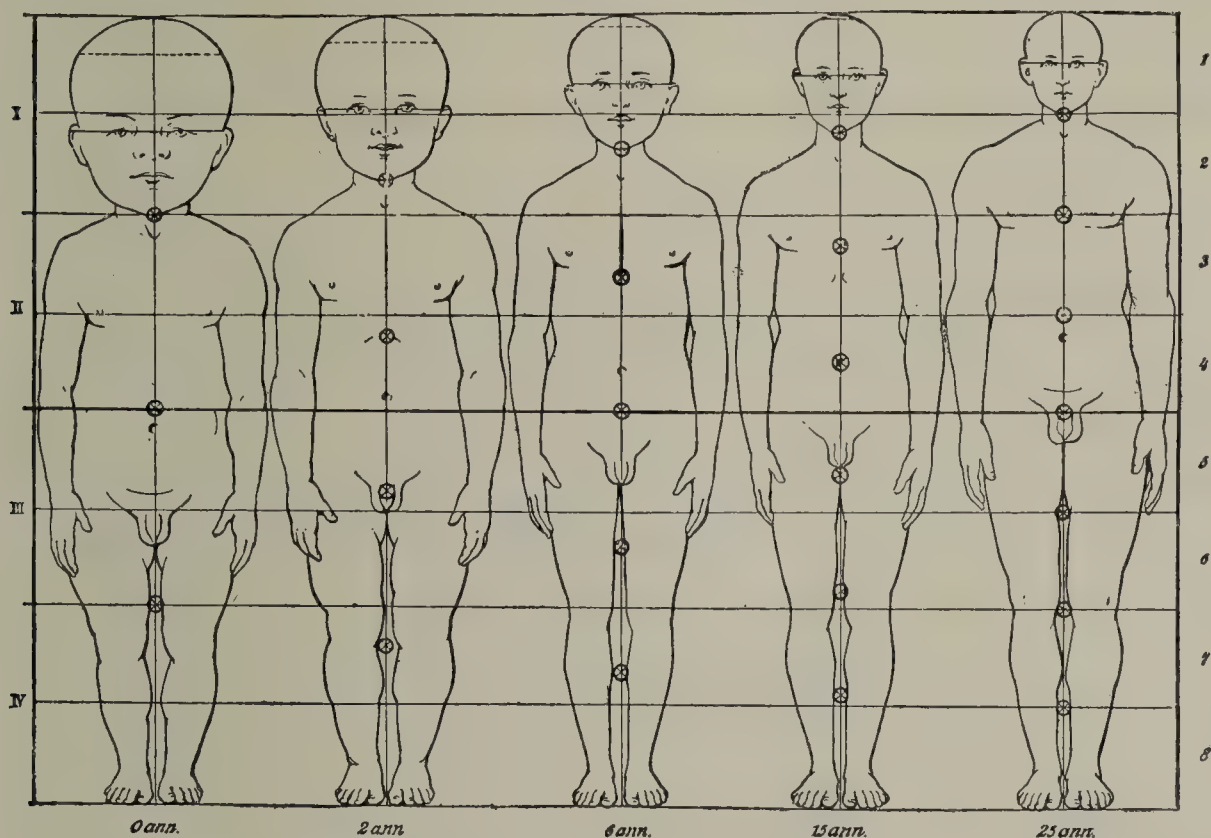


FIG. 25.—Relative proportions of the body at different ages (Stratz).

doses. If the case is one of athyria, the result of this treatment on growth and development is rapid and striking; and there are some cases in which stunted growth depends on defective thyroid action, although the children show no other characteristic sign of cretinism.

Premature Sexual Development.—The converse of infantilism is occasionally, though rarely, met with. It is found especially

¹ Lereboullet, *Les Cirrhoses Biliaires*, Paris, 1902, 76.

² Byrom Bramwell, *Scot. Med. and Surg. Journ.*, 1904, xiv., 321. (See p. 524.)

³ R. Miller and L. Parsons, *Brit. Journ. Child. Dis.*, 1912, ix., 289; A. E. Naish, *ibid.*, 337; D. Paterson, *Proc. R. Soc. Med.* (Children's Section), June 1920, 107; H. Morley Fletcher, *ibid.*, 118; and H. Barber, *Quart. Journ. Med.*, April 1921, 205.

in cases of tumour of the pineal gland, and in some forms of cortical hypernephromata (see pp. 521 and 522.)

When we compare the *proportions* of infants with those of older children and adults, we notice that their limbs are relatively short compared with the trunk, and that the circumference of the head and abdomen are large compared with that of the thorax. This is well shown in the accompanying diagram (Fig. 25) taken from Stratz's work, *Der Körper des Kindes* (Stuttgart, 1904). These differences gradually disappear owing to the different rates of growth of those parts.

Parents not infrequently suffer from unnecessary anxiety on account of a supposed enlargement of the head or belly of perfectly healthy children, owing to their overlooking these anatomical facts.

Development of the Sensory Functions and their Testing for Clinical Purposes.

Sight.—Very soon after birth a baby is able to distinguish light from darkness, but it is a long time before his eyes afford him much information. After the first few weeks most infants manifest distinct signs of pleasure when they are shown a shining object or a moderately bright light, and try to follow it. A restless baby may often be quieted for examination by being carried to a window. By the third month a normal child should show that to some extent he knows his mother—although it is probably her smile rather than her features that he recognises. By the sixth he should recognise many things; but for some time after he seems to know what they are he is unable to estimate their distance from him, and grasps at them when they are quite beyond his reach.

Bright colours please infants of even a few weeks old, probably only because of their brightness, but the power to discriminate between colours is comparatively long in developing. Red and yellow may rarely be recognised in the first twelve months, blue and green not generally till the close of the second, or during the third year.

In older children we *test the eyesight* by the same methods as are used in examining adults. In infants and young children thorough testing is of course impossible, but, with care, the main facts required can often be ascertained. To test a baby's

power of seeing we may watch whether his eyes follow a moving light, and if he seems to notice familiar sights unaccompanied by sounds. Thus, it may be observed at what distance he responds to his mother's smile, or recognises an approaching bottle at meal times; and, when he is older, the identification of such familiar and welcome objects as a penny or an orange forms a suitable test of vision.

It should also be noticed if the pupils contract with light, and, if the child is more than a few months old, on accommodation. It is sometimes interesting when testing the vision to bring the point of the finger suddenly close to the child's cornea without touching it, and observe whether this makes him wink. In normal babies under two months this manœuvre does not cause winking.

Although formerly indirect ophthalmoscopy was the method of choice in the child, the electric ophthalmoscope has rendered the direct method much easier and in consequence it is the one now usually employed. It is wise to have the pupils dilated as the child is apt to fix his eye on some near object, *e.g.* the light in the ophthalmoscope, and thus cause the pupil to contract. At times, and especially in the young infant, it is necessary to administer an anæsthetic for a satisfactory ophthalmoscopic examination. When this is done a mydriatic is essential, and a pair of eyelid dilators and a conjunctival forceps will be found useful to keep the eye open and to bring the pupil into view. Fundal changes are of great importance in the diagnosis of cerebral disease (choked disc) and congenital syphilis (choroiditis disseminata and pepper-ground retina). However, the frequent irregularity in the distribution of the choroid pigment in the normal infant must be remembered as it is apt to be mistaken for the results of disease.

As Dr Maddox first pointed out, the images produced by the *reflection of light on each cornea* are helpful in the examination of children's eyes in two ways. In the first place, by noticing the exact position of the image when the disc is in sight we may be assisted in finding it again without losing unnecessary time. Again, the comparison of the position of the corneal images in the two eyes may lead to the detection of a squint.

Photophobia is sometimes met with as a symptom of a general morbid condition. It is common during the onset of

measles and influenza, and it is also an early symptom of tuberculous meningitis and cerebral hyperæmia, and is characteristic of erythroedema or "pink disease" (p. 393). It is, of course, often due to local disease of the eye, and may in rare cases be caused by peripheral irritation from the mouth (p. 52).

Errors of Refraction.—If there is any suspicion that the eyesight is defective, the refraction should be examined, and any error present corrected, without delay. It is to be remembered that while *hypermetropia* and *astigmatism* are congenital defects, *myopia* is only acquired in later childhood, although the tendency to it is often hereditary. It is comparatively rare to find it before the sixth year, and it becomes increasingly common and severe during school life. A great deal can be done to prevent its increase by insisting on care in using the eyes, not holding the book too close or stooping over it, and not reading with insufficient light. These precautions may be relaxed as the child gets older, and are usually no longer necessary after twenty.

Recurrent Functional Micropsia.—This name may be given to a trivial disturbance of vision which is not very rare in older children. Its presence is usually only discovered by questioning, as it does not cause any great distress. Sometimes, however, if it recurs frequently and lasts long at a time, the annoyance it gives rise to is considerable; and the medical man may be consulted either on this account, or because it has suggested anxiety as to the condition of the child's eyes.

More or less suddenly the child finds that the furniture and other objects in the room appear much smaller than usual—as if they were three or four times as far away as they really are—although their details are as clear, or clearer, than before. After a few minutes this passes off, and it sometimes seems to go sooner if an attempt is made to focus the sight on a near object, such as the hand. While the micropsia lasts it interferes with reading.

There is no obvious cause for the onset of the symptom. Sometimes it comes on when the child awakens in the morning, sometimes in the evening when he is tired and sleepy, but it may begin at any time in the day; and it is not only when much eye-work is being done that it appears. We have known it to occur with a high degree of myopia, also with quite

normal vision; with chronic dyspepsia and with apparently perfect digestion. It is not associated with headache or giddiness.

The micropsia varies greatly in degree and in the frequency of its recurrence. It often begins about the tenth year, and ceases altogether between twenty and twenty-five, if not sooner; and it may cease temporarily for months or years during childhood or adolescence, and then recur without any obvious cause. The cause of the symptom is obscure, but it seems to depend on a passing relaxation of accommodation. It is apparently of no importance with regard either to the child's general health or to his vision, but probably it indicates a somewhat strained condition of the nervous system.

Blindness.—Want of sight, apart from any obvious disease in the eyes, occurs under various conditions in childhood. In infants apparent blindness, with a normal fundus and normal media, is a common indication of mental defect.

Temporary blindness often occurs in basic meningitis and chronic hydrocephalus, when the fluid is increasing. It may last for months, but usually recovers completely. We also occasionally see children who have become temporarily blind in the course of whooping-cough or uræmia, and especially after severe convulsive attacks accompanied by coma. In these the blindness may persist for weeks or months; the pupils are usually normal and active.¹

More than half of the cases of permanent blindness met with in infancy are due to purulent conjunctivitis. Many are the result of congenital malformations. The remainder have followed internal inflammation of the eyes—syphilitic or other—and a few are due to injuries. The possibility of congenital cataract must be borne in mind, as it is a condition in which operative interference may help. Unfortunately, however, congenital cataract is frequently accompanied by some serious brain lesion.

Congenital Word-Blindness or Visual Aphasia.²—In examining school children who have difficulty in learning to read it is important to remember the symptoms of congenital word-

¹ Ashby and Stephenson, *Rep. Soc. for Study of Dis. in Child.*, 1903, iii., 197.

² James Hinshelwood, *Congenital Word-Blindness* (Lewis), London, 1917.

blindness ; unless they are looked for, the child may be thought deficient in mind or eyesight and wrongly treated.

When this condition is well marked, the child, though he sees well and is quite intelligent, has difficulty in recognising printed letters or words, or in some cases, figures. The visual centre is all right, and so is that for the interpretation of what is seen, but the path between them has remained undeveloped. In many of the mild cases the patient can make out figures, and may know single letters when he sees them, but he cannot recognise even short words by sight. Thus he may know the picture of a cat at once and be quite able to name the letters c, a, and t, separately, and yet he will puzzle for an indefinite time over the word "cat" unless he is told to spell it aloud. When he does so, he at once names it correctly. He cannot take in the word at a glance, as a normal child does, but only sees the separate letters ; yet, when he names the letters, he knows at once what they spell. To put it otherwise, if the letters enter his head by the eyes, they remain separate and without meaning to him ; but, if by the ears, they go straight to where they are formed into words, and he appreciates their significance without difficulty. Such children have generally a good memory for sounds, and this must be taken advantage of in their training. They require careful special teaching, and with time and patience they can be greatly improved, even in bad cases.

Hearing.—During the first day or two of life all children are deaf ; but by the second week, if not earlier, they should be able to hear loud noises quite well. If a child pays no attention to a loud sound behind him by the fourth week, he is probably either deaf or mentally deficient. By about the end of the third month the child can usually recognise the direction of a noise and turn towards it. It is much longer, however, before children can distinguish between different sounds. Thus, among one hundred normal children, Demme found only two who seemed to recognise their mother's voice by three and a half months. Often they are much later in doing so. By six months, most babies like being sung to, and young infants generally seem pleased by jingling and rattling noises. If an infant on the bottle who seems in good health shows no interest when the sounds of the usual preparations for a meal are being made, although he cannot see them, it is probable that either his hearing or his intellect is at fault.

In examining infants' ears we must remember the shortness of the external meatus, which is such that a speculum is often scarcely necessary; and also the obliquity of the tympanic membrane. In testing a young child's hearing, the crumpling of paper is more useful than the ticking of a watch, as it has much pleasanter associations for him. The child should be placed so that he cannot see the examiner's lips, and made to repeat short words after him. Another simple way of testing the hearing in young children is to shake the closed fists simultaneously, one on either side of him—one of the hands being empty and the other containing a couple of coins. If he hears, the child will generally turn at once to the fist in which the coins jingle.

Hyperacusia.—An abnormal tendency to start at any sudden sound is not uncommon in the healthy infant. A sudden noise such as the explosion of fireworks or the striking up of a band may even call forth convulsions. This hyperacusia is, however, a common symptom in various forms of nervous instability. An exaggerated form of this is often met with in cases of spastic diplegia and in cerebro-spinal meningitis.

Deafness should, of course, always lead to an examination of the ears, and of the naso-pharynx for adenoids. Complete deafness from disease of the inner ear sometimes sets in during cerebro-spinal meningitis, also rarely in mumps and whooping-cough, and in the course of congenital syphilis in older children. It is important to bear in mind that if deafness sets in during early life—before the fifth year—the child usually and rapidly loses the power of speech until he becomes quite dumb. In such cases, unless a careful history is taken, the condition may be mistaken for congenital deaf-mutism, which is a definitely hereditary Mendelian characteristic. In all such cases the prognosis as to recovery of hearing is exceedingly bad. In deafness from otitis media the extreme importance of persistent carrying out of the treatment must always be impressed on the parents.

A lesser degree of *impaired hearing* is very common; and just because it is not complete and the child evidently hears loud sounds, it is apt to be neglected by the parents. Mental deficiency, by causing inattention to sounds, may make not only the parents, but often the physician as well, think that the disease is one of hearing rather than one of the mind. Such partial deafness interferes greatly with a child's school-work,

and, when it occurs early, with his learning to speak. If an older child "does not hear whispered speech with at least one ear at a distance of six feet, he cannot profitably remain in the ordinary classes of an elementary school."¹

Deafness does not usually cause during childhood any mental impairment or melancholia; in fact, deaf children often seem unduly alert, because the other senses are specially active, *e.g.* sight, touch, and smell. In this way the mental defective child and deaf child are in marked contrast.

Congenital Word-Deafness.²—This is a condition analogous to congenital word-blindness, but it is even less common. Children who suffer from it have good hearing and good sense. The higher centre which receives the sounds, and that which interprets them, are both normal, but the path between them is defective. When the child is spoken to, he pays no attention and is thought to be deaf till it is found that he is at once attracted by music, even when it is not loud, or by any household sound in which he is interested. Speech, in these cases, is naturally not acquired at the usual age, but it can be learned in time, although at first the child repeats the words and sentences after his teacher, as a parrot does, without attaching any meaning to them. The treatment consists in training the patient in lip-reading. He finds it comparatively easy to recognise words which he sees on the speaker's lips.

Both **Taste** and **Smell** are relatively well developed within the first few days of life. They do not, however, acquire a great deal of acuteness until later childhood, as is evidenced by the way in which infants take all sorts of obnoxious medicines with apparent relish. It is seldom necessary to examine them in children for clinical purposes. When it is, familiar articles of food form the best tests. Little children often show a liking for tastes which are unpleasant to adults.

The **sensibility** of the child to **touch**, **temperature**, and **pain** is not very acute at birth, but it soon increases. The testing of these sensations in young children demands infinite tact and patience, and often leads to little result. Painful sensations are usually the only ones that can be relied upon. It is always well to repeat the observations several times, on separate occasions, before being sure of their accuracy.

¹ J. Kerr Love, *Diseases of the Ear in School Children*, Bristol, 1919, 59.

² C. J. Thomas, *Child Study*, January 1904, i., No. 4, 97.

Thiemich has pointed out¹ that habitual presence of general analgesia (to pin-pricks) is characteristic of imbeciles even in babyhood, and that they often show a defect of taste.

Development of the Motor Functions.

The young infant has a fair amount of muscular force at his disposal, but his movements at first are mostly not voluntary. Some of them are *random* or *aimless*, being apparently the expression of a mere overflow of energy. Such are the grimacing, kicking, opening and shutting of the fists, and so on, which are so characteristic of the young baby. Others, like the blinking which follows a flash of light or a puff of wind, are simply *reflex*. The crying and sneezing of the child at birth are also of this nature.

Then there are movements which may be classed as *instinctive*. In these, reflex action plays a certain part, but they are also definitely purposive. The human baby at birth has very few of these actions except sucking. One of the most interesting of them is the firm way in which the hands close automatically on a finger or other object placed within their grasp. When the object grasped is gently raised, the child's instinctive grasp is so strong that he can be lifted right off the bed without his letting go.² This power of holding on is quite different from the voluntary grasping of older babies, and it is lost within a few days of birth. In young monkeys this instinctive grasp is much stronger and lasts far longer. In them it has a great practical value, for it enables them from the first to take such a firm grasp of the hair on their mother's chest that she is able to use all her limbs for running and climbing. The so-called "infantile form" of the plantar reflex (p. 681) is perhaps another movement of this kind.

As the baby's brain grows and develops, his random and instinctive movements become gradually stronger and he gains more and more control over some of them, so that in time they come to be done on purpose.

His power of firm *grasping* is slowly regained, and if he is over five months old the object grasped will probably also be carried towards his mouth. Should a child's fingers show

¹ *Deutsche Med. Wochenschr.*, 1900, xxvi., 34.

² L. Robinson, "Infantile Atavism," *Brit. Med. Journ.*, 1891, ii., 1226.

no inclination to close on an object placed in his palm, it is a morbid sign, suggesting usually either severe present illness, paralysis, or great mental impairment.

A baby is usually unable to *hold his head erect* until he is three or four months old, according to the degree of his muscular development.

The power *to sit up* appears at a time which varies with the vigour of the individual baby. A child, however, cannot generally sit, even for a short time, until he is six or eight months old, and then only if carefully propped up. Not until he is eleven or twelve months old does he permanently acquire the capacity for sitting unsupported.

Some children *creep* before they walk—as early, perhaps, as the ninth month; with others, creeping is a later accomplishment. A strong baby generally begins trying *to stand* by the ninth or tenth month, and may be able to do so for a little by the eleventh or twelfth. The exact time at which a child *walks alone* varies considerably in different instances—fourteen or fifteen months is perhaps the average age. Some are able to do so at nine months, and others not till seventeen or eighteen months. It is well to remember Dr Gee's axiom, though it is by no means absolute, that a child "who is not idiotic or weakened by some recent acute disease, and who cannot walk at eighteen months of age, is either rickety or paralysed."¹ Dr Dawson,² working in the Sick Children's Hospital, Glasgow, found that the intelligence of the child during later childhood was roughly proportionate to the age at which he had learned to walk.

Backwardness in holding up the head and in other muscular movements has a similar significance to delay in the power of walking. A healthy infant takes the keenest delight in the free exercise of his muscles, and any habitual disinclination to move is a morbid sign. If a young baby is swayed up and down in his nurse's arms he shows evident signs of pleasure, and his limbs move in time with her movements. If this does not occur, and her movements arouse no answering efforts on his part, there is reason to suspect the presence of an extreme degree of mental defect.

When a child who has been walking for some time "goes off

¹ "On Rickets," *St. Bart. Hosp. Rep.*, 1868, iv., 72.

² S. Dawson, "Intelligence and Disease," *Med. Res. Council Rep.*, No. 162, 1931, 49.

his feet," this is often a sign of advancing rickets. It may, however, be due to other general disturbances. In some children, for example, it occurs from time to time along with the appearance of uric acid crystals in the urine.

An interesting difference between the muscular action of very young infants and that of adults has been pointed out by Thiemich.¹ When an adult closes his fist forcibly the flexion of the fingers is accompanied by an associated dorsiflexion of the metacarpus. Similarly, a forward movement of the metacarpus goes with forcible extension of the fingers, and extension and flexion of the leg are associated, respectively, with plantar flexion and dorsiflexion of the foot. The co-ordination of these associated movements has been said to be of cortical origin. In the great majority of young infants, clenching the fist is associated with volar flexion of the metacarpus, and extension of the leg with dorsiflexion of the foot. The normal adult phenomenon only begins to be habitual in healthy children about the third or fourth month in the great majority of cases, although occasionally it is found earlier. In weakly children, even at a considerably later age (say seven months) there is dorsiflexion of the hand on seizing a pencil; but if it is pulled away from the child, his hand at once assumes the position of volar flexion characteristic of the pre-co-ordinate period of life. This phenomenon only occurs in weakly infants, and seems to indicate how easily co-ordination is exhausted in them.

Another peculiarity in the muscular system of very young babies, which should be borne in mind, is its *hypertonicity*.² A certain degree of this is always present in health. Sometimes, however, it is exaggerated to such an extent as to suggest a doubt as to whether the infant is not the subject of congenital spastic diplegia. It is due, apparently, to delayed development of the cortical inhibition, and has no morbid significance. It disappears entirely as the child grows older.

Sleep.

During the early weeks of life the infant should sleep nearly all the time that he is not being fed or washed, or having his clothes changed—perhaps 20 hours or more out

¹ *Zeitschr. f. klin. Med.*, 1902, xlv., 226.

² Hochsinger, *Wiener med. Wochenschr.*, 1900, L., No. 7, 314.

of the 24. As he grows older, his need for sleep becomes gradually less, but by the end of the first year he should still be allowed from 14 to 16 hours, and during the second and third years from 12 to 14 hours in the day. One or two of these hours are to be taken in the afternoon and the rest at night. From four to five, he should have 10 to 12 hours' sleep, all at night; and from six to ten years, 10 or 11 hours. For schoolboys and schoolgirls between twelve and sixteen, 9 hours' sleep should certainly be considered the minimum allowable; and most of them will probably benefit if they can

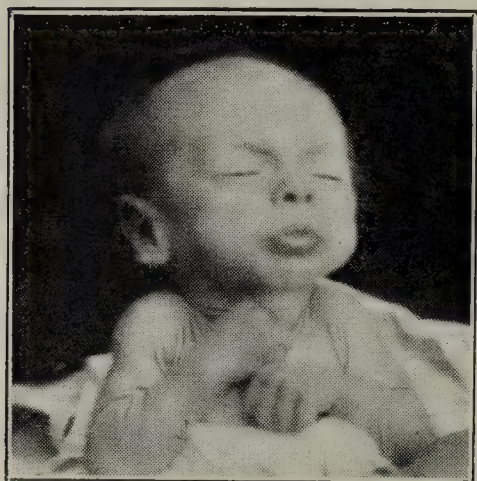


FIG. 26.—Atrophied infant of 7 weeks. Natural attitude of upper limbs during sleep, and imperfect closure of eyelids.

have $9\frac{1}{2}$ to 10 regularly.¹ In the light of physiological knowledge, the idea, still held by some parents and schoolmasters, that boys are made manly by curtailing their normal amount of sleep, can only be regarded as a foolish error. Insufficient sleep is a common cause of lowered mental energy and lessened nerve force, and is very apt in delicate boys to lead to a nervous breakdown.

The *attitude* the child assumes during sleep is worthy of notice.

A healthy infant usually lies on his side, or rather with his body semi-prone or prone and his head turned face downwards on the pillow. His limbs are generally fully flexed, so that the knees tend to touch the abdomen and his hands are held close up to his chin (Fig. 26). As Henoch pointed out, this attitude may be regarded as reminiscent of intra-uterine life. It is apt to be altered in disease, and therefore its presence in any case is a reassuring sign.

The eyes should be closed in healthy sleep. In severe acute illness, with collapse and rapid emaciation, the eyes of the sleeping child are often partially open. This is seen also in atrophy (Fig. 26) and in extreme debility from any cause, owing probably to a lack of tone in the orbicularis muscle. Incomplete closure of the eyes during sleep, however, is not necessarily a serious symptom. In nervous, irritable children

¹ See excellent paper, *On the Hours of Sleep at Public Schools*, by T. D. Acland, M.D. (Churchill), London, 1905.

—such as grind their teeth and have night-screaming attacks—this is frequently seen after an evening party; and it merely indicates that they have had an undigestible meal or too much excitement.

The mouth should also be closed. If it is kept open habitually during sleep, this suggests the probable presence of enlarged tonsils or adenoid growths. The sudden beginning of this symptom indicates rapid swelling in the nose or at the back of the throat, such as occurs in diphtheria and scarlet fever.

Young infants, after the first month or two of life, are easily disturbed; but after three or four years the child sleeps the enviably sound sleep which is so characteristic of healthy childhood, and is difficult to waken. The baby should be trained to go to sleep when left alone in bed, and not to expect to be rocked and sung to. He should, of course, always occupy a separate cot. The more sleep nervous children can be induced to take, at all periods of their lives, the better it is for them, and the more likely are they to grow out of their nervousness. The great importance of sleep to children with acute illness is not to be forgotten, and the patient must not be lightly wakened even for the purpose of feeding.

The disorders of sleep will be considered later (Chapter XXVIII., p. 692).

Speech.¹

Development of Speech.—A baby's first cries are merely instinctive, and have no intentional meaning. In time, however, he finds that his cry results in his being fed or otherwise attended to, and so he learns to cry when he wants anything. Later he gets to know that certain words or syllables mean certain things, and that by using them he obtains what he wants sooner than by indiscriminate crying.

By the time he is twelve months old he knows the meaning of a good many words, and there are usually one or two articulate sounds which he uses with a definite meaning. During the second year his knowledge of words increases fast, and he can frame short phrases before the end of it. The age at which different children begin to talk varies, however, greatly, and often it is impossible to tell why it does so.

¹ For the best account of speech, normal and abnormal, the reader is referred to Prof. John Wyllie's book on *The Disorders of Speech*, Edinburgh, 1894.

Dawson,¹ working in the Royal Hospital for Sick Children, Glasgow, on the intelligence of children, found that the age at which the child commenced to speak was directly proportionate to his mental intelligence during later childhood. This we have already remarked was also true, but not to the same extent, with regard to walking. This is easily understood, as talking is a more intellectual process than walking.

During the time that they are learning to speak normal children almost always make use of some words of their own invention; but as their powers of talking develop these baby-words are forgotten. Similar words are often invented by imbeciles, and in them they may continue to be used during life.

Some children, however, never use baby-talk; there is no doubt that the habit of parents of employing such language themselves when talking to their infants is in great part responsible for its use.

Backwardness in learning to speak is a thing which naturally causes anxiety to parents. It may be explained in a number of ways:—

1. It is often attributed to *tongue-tie*. This is a mistake, for while this small abnormality may interfere slightly with sucking, and possibly, as the child gets older, with the pronunciation of certain letters, it never delays speech.

2. In many cases it is a symptom of *mental defect*; and late speech always suggests the possibility of mental backwardness.

3. Not uncommonly, slowness in acquiring speech is due to *defective hearing*. This cause is apt to be overlooked, owing to the child responding at once to loud noises, although he is sufficiently deaf to have a difficulty in noticing and imitating the lesser variations of sound which make up ordinary speech.

4. It may also be due to *general backwardness in development* following severe illness, or accompanying some such weakening condition as rickets or chronic diarrhoea, and words come as the child gets older and stronger.

5. Some children with normal intelligence and good hearing, and who have not been recently ill, are long in talking, and it may be impossible to tell why they do not talk sooner. The main practical point is that in such cases the prognosis is

¹ S. Dawson, "Intelligence and Disease," *Med. Res. Council Rep.*, No. 162, 1931, 49.

generally good ; and that slowness in beginning to speak is not in itself a proof of defective intelligence.

6. We must also remember the possible occurrence of *congenital word-deafness* which is apt to be mistaken for absence of hearing (p. 34).

Aphasia may occur in children as in adults, as the result of organic brain disease (for example, embolism or encephalitis) ; it may, however, be purely functional. In the former case, it is generally, although not always, the left hemisphere that is affected. In the latter, it is most frequently seen during recovery from one of the infectious diseases, such as enteric fever, whooping-cough, or measles, or in connection with an attack of chorea.

Nasal speech, when it is habitual, is generally the result of enlarged tonsils or adenoids, if not of a cleft or ulcerated palate. When it sets in suddenly it may be due to inflammatory swelling of the parts at the back of the throat, but it should always suggest examination for diphtheritic paralysis of the soft palate.

Stuttering or **stammering** is sometimes met with as early as two years old in neurotic children. It seems as if the child's ideas come too quickly for their co-ordinate expression. In these cases it is often temporary, but it is apt to return in later childhood. Persistent stuttering generally begins after the commencement of the second dentition. It is commoner in boys than in girls. In some cases, probably only in children who are predisposed, it is easily acquired by imitation.

When young children stutter, little or nothing can be done in the way of direct treatment. Attention should, however, be given to the general health, all sources of nervous excitement and irritation avoided, and singing and shouting encouraged as much as possible.

In older children great improvement follows careful training.¹ The child must attend to the way in which he speaks, and the mechanism of speech must be explained to him, so far as he is capable of understanding it. He must be taught to speak *with voice*—that is to say, with a certain musical resonance ; he must also take breath frequently while speaking, so as never to speak from an empty chest. If capable of singing, he should

¹ Full directions will be found in Prof. Wyllie's book ; also in Dr H. G. Langwill's paper on "Stammering and its Treatment by the General Practitioner," *The Practitioner*, Jan. 1903.

exercise his voice in this way regularly, and he should practise for fifteen minutes at least every day, reading special sentences containing the sounds he has difficulty with, as well as ordinary poetry and prose. The child's general health must also be attended to, and special care taken that he is not being over-worked at school.

Lisping is common in a temporary form in little children. It consists in a difficulty in pronouncing some of the consonants such as *s*, *k*, *th*, and *r*, which become *th*, *t*, *s* or *f*, and *w*, respectively. In some it persists for a long time, and it may be very severe. The more aggravated forms are sometimes spoken of as *Lalling*. This condition is often met with in mentally defective children. When there is no defect of hearing, according to Ashby,¹ "marked slurring or baby language in a child of five or six years of age is almost always associated with subnormal intelligence."

Idioglossia is a term given by Hadden² to a severe form of lalling. In it the patient habitually substitutes certain consonants which he finds easy to say (usually *t*, *d*, or *n*) for all those with which he finds difficulty. This makes his speech sound like an unknown tongue, till it comes to be analysed. The condition is commoner in boys than in girls; and the patients are often very bright children. The peculiarity shows itself when the child begins to speak; and by the time he is about four years old, if not sooner, the mother seeks advice.

The *treatment* of these speech-defects consists in training the child in articulation, as deaf-mutes are trained, by the oral method. The child is placed before the teacher, who demonstrates to him, by exaggerated movements of his own lips, tongue, and larynx, the way in which the desired sounds are produced. The child is also made to practise regularly simple exercises which contain the sounds which he has most difficulty in pronouncing. It is also particularly important that he should be separated from those who understand his peculiar jargon, so that he may be induced to take trouble to make himself intelligible.³

¹ *Med. Chronicle*, Oct. 1903, 1.

² *Journ. of Mental Science*, Jan. 1891, xxxvii., 96; see also W. S. Colman on "Impediments of Speech," *Allbutt and Rolleston's System of Medicine*, 1910, viii., 576.

³ The various Education Authorities supply facilities for training in the case of speech-defects.

While special treatment greatly accelerates the cure in these cases, it must be remembered that even without it, if the child's intellect is normal, there is a strong tendency to recovery. We have watched several children whose speech was badly affected in this way when they were four or five, and yet became practically normal by the time they were eight or nine, although they had had no treatment except that which the home and the Board School had supplied.

The Home Care and Treatment of Physically Defective Infants and Young Children.

All medical men in active practice meet from time to time with what, for want of a better name, we may call *Physically Defective Children*. This term includes those who, owing either to congenital defect or disease, or to past infantile ailments of a serious nature, have been permanently damaged so as to be more or less handicapped for life—although very often their original trouble no longer calls for any active medical or surgical treatment.

These children belong to three classes: the *blind*, the *deaf*, and those who are *crippled* or *invalids* from other causes. They have important requirements beyond those of the other children we are in the habit of looking after from day to day. Like the others they have, of course, illnesses to be treated and pains to be relieved; but what, in view of their future, they need far more than anything else is that they should be properly trained from the first, so that, in spite of their defect, they may have a chance of growing up as like other children as possible in their capacity and behaviour. Without such special care they are certain to fall increasingly behind their fellows in health and happiness and to make much less of their lives than they ought to have been able to do.

Their education, when they reach the school age, has of course very often to be carried on in special institutions or schools by specially trained teachers. In their earlier years, however, at the time when Nature is inspiring them with a keen desire to learn and practise the use of all the senses they have, their upbringing is *entirely in their mother's hands*, and she only is in a position to train and encourage them to overcome their difficulties and learn to behave, as far as may be, like other children.

Our duty, therefore, as medical men is to see to it that the child's mother understands what she can and should do for him; and, especially, to arrange that the home shall be visited regularly by a competent district nurse or other visitor, who is able to instruct and supervise her until she has become thoroughly familiar with her duties to the child.

When thorough training of this kind is carried out, it makes a great difference to the child's present contentment as well as to his growth in body and mind, to his ability to benefit from education later, and also to his mother's peace of mind.

The mother must, therefore, be shown how to lead the baby on gradually to make the most of those powers which he has, in order that they may as far as possible make up for those he has lost. She must also pay particular attention to the details of his conduct and manners, and check at once any bad habits he begins to form. She must, especially, do all she can to make him *self-dependent*, by encouraging him in everything he is able to do; and never allowing anything, however small, to be done for him if he can be induced to do it for himself.

Blind Children.—The parents of the blind baby must always remember that although his sight is defective, he is not otherwise an abnormal child, and that they must be careful to do nothing to make him so. He must not be indulged more than the others, and should always be treated, as far as possible, as if he were able to see; and he must be constantly stimulated to self-dependence.¹

His having lost his sight makes it necessary that his other senses should be carefully cultivated. The wonderful acuteness of hearing and touch which blind people often show is not a free gift of Nature; it is simply the outcome of endless painstaking practice. To this, therefore, the child must be constantly encouraged. It has been found that, when blind children who are mentally normal are neglected during their infancy, they are usually unable to use either their hearing or their sense of touch nearly as well as normal children of their age; and by the time they are four or five years old are generally at least two years behind their fellows. They are also often babyish, and uncleanly in their habits. If properly

¹ A useful leaflet of *Suggestions to the Parents and Friends of Blind Children* can be obtained at the Central Office of the Charity Organisation Society, Denison House, Vauxhall Road, London, S.W. 1.

looked after at home, however, the blind child will grow up nearly as capable as other children, and will be little if at all behind them in behaviour and intelligence.

When he is able to creep, the blind baby should be allowed to move about everywhere as freely as is consistent with safety and to find the things he wants for himself. In this way he learns by experience, from the knocks he gets, how to take care of himself.

Toys are a valuable source of instruction, and do much to train the mind as well as the hands. Such occupations as the threading of small beads are particularly good for him. They help to develop the sensitiveness of his finger-tips in a way that will be useful later when he comes to learn the Braille alphabet.

He should be allowed to handle and investigate all sorts of objects; and knives, pins, and other things that will hurt him, if he is careless, must not be altogether kept out of his way.

From the first, attention must be given to making the child cleanly and regular in all his ways, and tidy in his person and habits. As he grows older, he must be taught to feed, wash, and dress himself; and he should be used to take his meals with the rest of the family, and be talked to just like the others.

Blind babies are more prone to bad habits of many kinds than any others, except some of the mentally defective, so that their mothers must be warned to look out for and check such practices whenever they begin. Among the commonest of these are nodding and shaking the head, swaying the body backwards and forwards, eating dirt of all kinds, and masturbation. One dangerous habit which is peculiar to blind babies consists in rubbing the eyes vigorously with the knuckles. This is sometimes practised with such force as to burst the eyeball if it has been weakened by disease. The attraction of all these bad habits is lessened greatly when the child has plenty of open-air exercise and occupation, and by everything that increases his interest in life.

As the children grow older, they are apt to acquire an awkward gait—either holding the head forward or stretching it back when they walk. Such postures must be watched for from the first and corrected, or they will be a serious drawback in after-life.

Deaf Children.—When a baby is only partially deaf it is important that he should always be spoken to in a loud voice,

so that he may get into *the habit of trying to hear*. Otherwise he will be very long in recognising that speech is of any importance, and will therefore cease to listen to it.

In the great majority of deaf babies the defect of hearing is so great that they cannot learn anything at all from spoken words—although all congenital deaf-mutes, and many others, are able to perceive very loud sounds, either by hearing or by feeling in some way the vibration they produce. It is, therefore, of great importance that they should as soon as possible be taught to watch their mother's lips when she speaks to them.

To understand what the loss of hearing means to a baby we must realise how very much of his first knowledge of life naturally reaches his mind through his ears. Sounds mean little to him during the early months, but as they are repeated and repeated, the meaning of some of them gradually dawns on him; and, before he is a year old, he understands a great deal of what is said in his presence. In this way he learns many things which please him, and much also which influences his conduct. In time he comes to realise the advantage of words and to try to use some himself.

By his want of hearing, therefore, the deaf baby is placed at a serious disadvantage in gaining a knowledge of life, and, unless pains are taken to give a special training to his sight during infancy, he will, in addition to not being able to speak, be far behind other children of his age in many other ways when he reaches school age. It is said that a deaf child who has had no special training is usually no further on, in many respects, at seven than a normal baby of three.

The essential part of the deaf child's teaching consists in his being trained to *see* and recognise on his mother's lips the words which his defect makes him unable to hear. The power to do this can only, of course, be very slowly acquired, and learning it is a laborious business for the child as well as for his mother; but experience has shown that, with patience, it usually succeeds to a surprising degree.

The key to success in this matter lies in the application of the plan of endless repetition, which all good mothers instinctively use in training their normal infants to understand them and in time to speak. The constant repetition of simple words all through the day not only brings the child early to

understand the importance of language, but has further a valuable and increasing effect in stimulating his intelligence. It is found that normal babies, who for any reason are not much spoken to, are generally much slower than usual in their mental development.

In her excellent and most helpful little pamphlet, *What the Mother of a Young Deaf Child can do*,¹ Miss Martin gives the following rules to be observed by the mother in her first attempts at teaching her child to read her lips:—

“Every time you speak to your child,

“1. Get down so that your mouth is on a level with his eyes.

“2. Always turn your face so that the light may fall on your mouth.

“3. Always bring the child's eyes to your mouth, and speak *distinctly but without exaggeration*.

“4. Speak naturally and try to realise that you are doing quite *a natural thing* in teaching your child to lip-read.”

The mother must get into a habit of *never* speaking to the child when his back is turned, or when his eyes are not looking at her mouth, and all exaggerated speaking must be avoided.

It will of course take a long time and much perseverance before the baby learns always to watch his mother's lips; but, when he has learned this, his education will begin to go ahead. In Miss Martin's little book simple directions and suggestions will be found as to many other important details of the deaf child's treatment. It is a very great advantage to the mother also to have regular visits from some one who is able to guide and encourage her in her child's training.

The deaf child is usually too observant to learn the awkward habits that the blind are liable to. His chief morbid tendency is towards getting self-centred and irritable. The best remedy for this is mixing freely with normal children who are friendly and do not tease him.

When the child reaches school age he will be taken care of by the Education Authorities, to whom the parents must apply. But until he reaches school age all his education must be at home, and this cannot be commenced early enough. Some

¹ To be obtained from Messrs Hill & Ainsworth, Ltd., Glebe Street, Stoke-on-Trent. Price, post paid, 3d.

Institutions, *e.g.* Donaldson's Institute, Edinburgh, take children over three years, whereas Langside Institution, Glasgow, only takes children over four years. Both these Institutions are under the Education Authority.

Cripples and other Chronic Invalids.—This group would be a large one if we were dealing with older children, but among those in the first few years of life there are not very many who belong to it. It includes those whose bodily activities are restricted more or less permanently by congenital malformations or defects of growth, and some who have been seriously damaged by disease in early infancy. We have, for example, cases of congenital heart disease, of spina bifida and other congenital forms of paralysis unaccompanied by mental defect, and various kinds of dwarfing and other deformities. Those whose debility is secondary to infantile disease are such as have been crippled by acute poliomyelitis, severe tuberculous lesions, or rickets.

Blindness and deafness, as we have seen, cause a delay in the infant's mental development. In many invalid babies another of the "gateways of knowledge" is more or less interfered with—that which leads to the brain through tactile sensations, muscular sense, and muscular activity.

The cripple baby's inability to get about, and to use his feeble or stiffened limbs freely, usually makes him longer than he should be in gaining experience in such matters as are normally learned by creeping round the room and taking hold of all sorts of things and handling them as other babies do.

In regard to the treatment of such weakly and invalid children, one need only emphasise again how essential it is to their happiness, and also to their mental development and bodily health, that they should not, by a lack of proper training in infancy, be rendered self-centred, discontented, and dependent.

CHAPTER III

THE TEETH

Dentition

The Temporary Teeth

THE temporary or milk teeth are 20 in number, and the average ages at which they appear may be given as follows:—

- | | |
|--|----------------|
| (1) Lower central incisors | 5 to 10 months |
| (2) Upper central and lateral incisors | 8 to 12 „ |
| (3) Lower lateral incisors and lower and upper
first molars | 12 to 14 „ |
| (4) Lower and upper canines | 16 to 22 „ |
| (5) Lower and upper second molars | 24 to 30 „ |

In normal children the teeth usually come in pairs—a tooth on one side cutting the gum about the same time as the corresponding one on the other. Those in the lower jaw almost invariably appear a little earlier than the corresponding teeth above, except in the case of the lateral incisors. The order in which they appear in healthy infants is generally much the same, but the intervals between the cutting of the different groups of teeth often vary greatly. Irregularity in the order of their eruption and their appearance singly, instead of in pairs, is characteristic of rickets and mongolism. Sometimes the first teeth appear at three or four months old; and, in rare cases, infants are born with teeth—usually the lower central incisors.

Delayed dentition may occur in apparently perfect health, the first incisors not showing till the child is ten, twelve, or even as much as fourteen months old. Generally, however, such delay indicates disease; and any child that has no teeth at ten months should be suspected of being rickety. It is better when the teeth do not appear through the gums too early; for, when they do so, they are apt to have thin enamel, and not to last so long as they might have done had their crowns had longer time to mature under the gum.

Symptoms of Teething.—In the majority of cases nothing unusual is noticed in the child's general condition while he is cutting his teeth, and there is little or no local disturbance, and it is only as a last resort that a diagnosis of "teething" should be made. Jacobi¹ in 1862 published a well-known book on *Dentition and its Derangements*, not so much to record the manifestations of teething as to focus attention on what diseased conditions it could not produce. It is, however, a matter of common observation that many babies are more or less out of sorts for a short time before a coming tooth appears, and are greatly relieved when the process is completed. The disturbance may be local or general, or both.

Local pain in the jaw is sometimes evident. A teething baby keeps his lips compressed and resents any attempt to look at his gums. He puts his fingers into his mouth as if it were uneasy, or he may screw up his face or put his hand up to his ear or jaw, as if visited by a sudden twinge of pain. A closer examination sometimes shows that the saliva is greatly increased in amount, that the gum over the coming tooth and in its neighbourhood is bright red, and the adjacent glands may be slightly swollen and tender. Rarely, a hæmorrhagic cyst may form over the erupting tooth.

The child is often flushed and feverish, whether the local disturbance is noticeable or not; and there may be restlessness, loss of sleep, and irritability. The appetite may also be lost for the time, and the bowels become constipated, or there may be slight diarrhœa or vomiting. Often the child loses weight or ceases to gain it. Less frequently, slight temporary neuroses develop; for example, there may be a tendency to winking the eyes, a frequently recurring cough, or a decided acceleration of breathing without any pulmonary disease.

Some children have, while teething, a tendency to certain diseases to which they are not subject in the same degree at other times. Thus it is not an uncommon thing to find a child who, with the appearance of each new group of teeth, has an attack of diarrhœa or vomiting, or bronchitis, which resists treatment while the teeth are in process of appearing, but rapidly recovers (under otherwise similar conditions) when they are through. Similarly, we see babies with eczema who have a marked relapse with each new group of teeth; and often an

¹ *Dentition and its Derangements*, New York, 1862.

obstinate eruption will disappear spontaneously whenever all the teeth have pierced the gum. In infants who are subject to convulsions or laryngismus the attacks are apt to recur and to be more severe during active dentition.

The Place of Dentition as a Factor in the Causation of Disease.—Teething used to be regarded as a frequent cause of serious and fatal disease. This is a dangerous belief, because there is usually associated with it the idea that, as teething is a natural process, the diseases accompanying it are to be tolerated and not checked as they would be under other circumstances. Consequently, we have often met with children exhausted with diarrhœa which has been allowed to go on untreated for weeks because it was held to be “only from the teeth.” The diagnosis of teething as the cause of any illness will always be a popular one, because it casts no blame on the parents, as exposure to cold, improper feeding, and rickets are apt to do.

A number of writers, in Germany and America chiefly, have gone to the opposite extreme, holding that, as dentition is a physiological process, it cannot cause disease—that “teething produces teeth, and nothing else.”

We shall be nearer the truth if we avoid both extremes. There can be no doubt that teething is not in itself a cause of death, and that its influence in producing and predisposing to disease used to be enormously exaggerated. At the same time it is equally certain that teething, like menstruation, pregnancy, and other natural processes which induce a degree of lessened stability, is often accompanied by considerable distress, both local and general, and like them may produce temporarily a tendency to disease which is not present at other times.

The effect of teething on a child's health varies with his strength, and especially with the state of his nervous system. While most children, fortunately, cut their teeth without anything but a slight passing indisposition, if even that, others who are rickety, neurotic, or otherwise weakly, may show signs of more or less severe reflex as well as local irritation. The symptoms which arise in this way may be alarming, not because they indicate a danger to life, but because they often lead us to suspect the presence of some serious disease. When we remember the amount of general disturbance which may occur in older children from phimosis or adenoids, or by the presence

of worms or scybala in the bowel, we can scarcely wonder that alarming symptoms are occasionally set up in babies by the irritation that accompanies dentition.

Severe Nervous Symptoms from Teething.—In very rare instances the process of teething has a profoundly disturbing effect on the child's nervous system and severe functional neuroses are set up. These may take the form of convulsions which recur with the appearance of each succeeding tooth. Occasionally there are a series of nervous symptoms. There may, for example, be violent, apparently causeless, screaming, intense photophobia like that from a corneal ulcer (although the eyes are normal), obstinate insomnia, giddiness, head-retraction or nervous vomiting. The symptoms set in suddenly when a tooth, or set of teeth, is appearing; they often last for weeks, and pass off abruptly when the teething is over for the time; and they usually recur when the next set of teeth is being cut. When such symptoms occur there is usually a noticeable absence of salivation. Cases of this type are very seldom met with, and the symptoms are apt to be attributed to much more serious causes. They require special treatment.

Diagnosis of Symptoms due to Teething.—It is easy to ascertain whether teething is proceeding or not, but often difficult to make sure of the part, if any, which it plays in producing such symptoms as are present. For example, a case of simple bronchitis may be mistaken for one of pneumonia on account of a high temperature and rapidity of breathing which are really due to the teething. In such a case, a careful consideration of the physical signs and the absence of real dyspnœa will help in deciding the diagnosis. Again, children who are suffering from dyspepsia and teething at the same time may be drowsy, irritable, and feverish; they may even have head-retraction and they often have vomiting, so that a mistaken diagnosis of early meningitis is possible. The absence of cerebral breathing and of a slow, contracted, and irregular pulse, of obstinate constipation, or of a retracted abdomen is in favour of the less serious condition, and should incline us to suspend our judgment until further symptoms appear. A knowledge of the physiognomy of cerebral disease will be found of practical value; and a timely dose of castor oil may remove diagnostic difficulties to a surprising degree.

We should not, therefore, be ashamed of diagnosing teething

as the cause of certain morbid phenomena in infants. This diagnosis, however (like that of hysteria in older patients), should never be made until all other causes of the symptoms have been carefully excluded, and we should not be satisfied with it until the child is well.

Treatment.—The restlessness, wakefulness, and irritability of teething children are often greatly relieved by the administration of a sedative such as antipyrine (1 to 2 gr.), phenacetin (1 gr.), or bromide of potash or soda (2 to 4 gr.). Preparations of opium, including the so-called “Soothing Syrups,” should never be used for this purpose in ordinary cases of teething. In the case of severe nervous symptoms, however, such as the violent screaming attacks referred to above, the administration of large and, if necessary, increasing doses of opium form the best treatment. When the teeth appear in these cases there is no difficulty in stopping the drug.¹ As already mentioned, an aperient such as castor oil frequently produces a striking improvement in the symptoms. Diseases which occur during dentition should be treated as at other times, and the fact that teething is proceeding affords no reason why treatment should be delayed or considered unnecessary.

Lancing the gums was at one time advised in all cases of obscure disease occurring at the age of dentition. It is now known that such a practice is not only unnecessary but probably hurtful in all but a few cases. When serious disease is present it is extremely doubtful whether lancing the gums can ever favourably influence its course. In cases, however, in which the gum is swollen, red, and tense over a coming tooth, and the child is suffering local pain, or showing signs of reflex nervous disturbance, lancing of the inflamed gum certainly often affords immediate relief, and can do no harm.

The Permanent Teeth.

The permanent teeth number 32. The order and time of their appearance are usually given as follows:—

First molars	6 years
Incisors	7 to 8 „
Premolars	10 „ 11 „
Canines	10 „ 11 „
Second molars	12 „ 13 „
Third molars (wisdom teeth)	17 „ 25 „

¹ J. Thomson, *Edin. Med. Journ.*, 1917, N.S. xviii., 203.

The first molars appear behind the second temporary molars, while the permanent incisors, canines, and bicuspid take the places of the temporary incisors, canines, and molars. The eruption of the permanent teeth is not a usual source of irritation, either general or local, in childhood, though the wisdom teeth, especially those of the lower jaw, may cause a surprising amount of distress when they are appearing. Delay in the appearance of the permanent teeth is one of the symptoms of sporadic cretinism, and is a useful help in the diagnosis of slight and atypical cases of that disease. In sporadic cretins who have been on thyroid treatment since infancy the permanent teeth often appear unusually early.

Caries of the Teeth.¹

Among civilised peoples, no other single disease of the body is nearly so common as dental caries; almost everyone has it. It usually begins in early life, and observations on school children before the War showed that it was rare to find one in twenty with perfectly healthy teeth. Since the War, in districts where dental school clinics have been active, matters are much improved, but generally dental caries is still very prevalent, with only slight differences, all over Great Britain, indeed in all civilised countries, and in every rank of society. Among uncivilised races dental caries is much less common; its prevalence among them varies from 20·8 per cent. among some negroes to only 1·4 per cent. among the Esquimaux.

Since the extraordinarily wide prevalence of the disease among us has long been known, the wonder is that our profession has shown, until recent years, so little active interest in searching out its causation and learning how to prevent it. It might be thought that this neglect was due to the ailment being comparatively harmless, but this is very far from being the case. Carious teeth not only lead to toothache, neuralgia, and disfigurement—not to speak of dentists' bills—but they are also occasionally a source of greater evils. For, if the disease is extensive, it frequently interferes with mastication.

¹ The authors are much indebted to the writings of Dr J. Sim Wallace, *The Prevention of Dental Caries*, 2nd ed., London, 1912; *The Teeth and Health*, London, 1926; *Oral Hygiene*, 2nd ed., London, 1929; to H. P. Pickerill, *The Prevention of Dental Caries and Oral Sepsis*, London, 1912; and to J. H. Gibbs, *Edin. Med. Journ.*, June 1917, 433.

tion to such an extent that the child suffers severely from chronic indigestion and consequent malnutrition; and, as we all know, even a single diseased tooth that has been overlooked may cause abscess formation in the gums and set up chronic septic absorption and keep a child anæmic and cachectic for years. Within recent years, however, certain aspects of the etiology of dental decay and the ways in which we can aid Nature in preventing it, have been thoroughly investigated by able members of the dental profession; and it is certainly time that we recognised and availed ourselves of their valuable contribution to preventive medicine.

They tell us that dental caries is not an inevitable evil and that it is not hereditary, but acquired; and they give it as their opinion that *few diseases are so easily and certainly preventable, provided simple and rational precautions are taken*. They would have us believe that caries of the teeth is a quite unnecessary malady of civilisation; and that we go on giving it to our children simply because we will not stop feeding them in foolish and unphysiological ways that frustrate Nature's plans for keeping the teeth healthy.

The Causes of Dental Caries.

The consensus of dental opinion ascribes caries of the teeth to the action of lactic acid produced by the fermentation of carbohydrates generally, but especially of sugar, which has become lodged and remains in crevices in or between the teeth. Such a state of matters is therefore intimately related to the type of food consumed. Anything which favours the deposition of these fermentable types of food predisposes to caries, and anything which precludes this happening is inimical to caries. The seats of election of caries—the irregular crowns of the molars and the teeth of the upper jaw in preference to those of the lower jaw—strongly support such a view. In the words of Sim Wallace, the whole matter is one of oral hygiene.

It is the processes associated with mastication which cleanse the teeth, and hence, as above stated, the matter is in great part one of diet. The call for thorough mastication of the food acts in several directions. In the first place it develops the muscles and jaws and thus more room is provided for the teeth, so that they are not overcrowded and badly placed. But mastication

also ensures the passage of the teeth through the bolus and the frequent movement of the cheeks, lips, and tongue against the teeth, both of which actions exert a definitely cleansing function; and finally, vigorous chewing stimulates the flow of saliva, which helps to wash away any debris which may be left behind. Hence crisp, spongy, and fibrous articles are advantageous, and the condition of the teeth when such food is consumed is in contrast to that following the use of sloppy foods such as custard, porridge, and pudding.

Within recent years an attempt has been made to associate dental caries with the absence of an accessory food factor. Mrs Mellanby,¹ on the strength of experiments in puppies, states that absence of vitamin D from the diet causes hypoplasia of the enamel of the teeth, and she postulates that this occurring in children will predispose to caries. No doubt hypoplasia of the enamel will render a tooth more vulnerable, but the distribution of dental caries is a serious objection to the adoption of any such theory regarding its cause. It is a well-known fact that the temporary teeth are more subject to caries than the permanent teeth, and yet it is the latter which are more frequently the seat of hypoplasia. And, further, the extreme susceptibility of the molars, which are richer in lime than the incisors, and of the teeth of the upper jaw in contrast to those of the lower jaw, would seem to definitely preclude any nutritional factor when it is remembered that the corresponding teeth of both upper and lower jaws are practically synchronous in their development.

Predisposing Causes of Caries.

(a) *Abnormalities of Position and Structure of the Teeth.*—When the teeth are well formed, regularly placed in the jaws, and used as vigorously as they ought to be, there is little tendency for fragments of food to be left adhering to them; but such matters are sure to gather and ferment when there are holes and corners anywhere. These cavities may consist of abnormal pits and fissures which have developed in the teeth themselves, owing to some such weakening disease as an infectious fever, dyspepsia, or whooping-cough having occurred at the time when the enamel at that level on the tooth was being formed. Much

¹ "Diet and the Teeth," *Med. Res. Council Rep.*, Nos. 140 (1929) and 153 (1930).

more frequently, however, the traps which catch and harbour the starchy and saccharine particles consist of crevices formed between adjoining teeth which are irregularly placed and crowded together. This irregularity and crowding arise often from defective growth of the jaws, which again is mainly due, in all probability, to too little use at an earlier period of life. Deficient development of the jaws from want of exercise may begin very early, for we find that they grow less normally in bottle-fed babies than in those on the breast; and the same thing happens in older children when they are fed only on soft pappy foods and given no tough and fibrous materials to practise them in the art of chewing. Defective growth of the jaws may also be one of the results of rickets, though rachitic children are no more subject to caries than non-rachitic children. Nasopharyngeal obstruction from adenoids and the injudicious and premature extraction of temporary teeth may also influence the form of the jaw. As we have already seen, the teeth of children who have suffered from severe chronic illness—such as rickets—in infancy are especially prone to decay, and the same is true of teeth which have been cut prematurely before the enamel has been properly formed.

(b) *Functional Defects of Salivation and Mastication.*—Anything which lessens the amount or changes the normal composition of the saliva is apt to increase the liability of the teeth to decay; and whatever discourages the free play of the jaws in mastication does likewise. A painful state of the mouth from bad teeth or any other cause has this effect.

(c) *Faulty Food and Ways of Feeding.*—Another very important factor in the causation of dental caries is the habitual feeding of young children on soft food with too much sugar in it, and too little sapid substances of other kinds. The giving of too little fruit, or fruit at wrong times, of too much farinaceous and too little animal food, are probably all dietetic errors which are bad for the teeth.

Diet in Infancy and Childhood in relation to the Prevention of Caries.—The solution of the problem of the prevention of dental decay lies then largely in the selection of a natural diet, and in proper habits of eating. Before the appearance of the teeth, the chief thing, of course, is that the baby should be on the breast, and if this is impossible, that he should be fed

on a diet which will not predispose to rickets, and in a manner as nearly as possible like breast-feeding.

When the teeth begin to appear, there are two other things that are important. The first of these concerns the consistence of the meal. When farinaceous food is given, it should not be mixed with the milk in a pulpy and relatively tasteless mass, but a certain amount of it should be taken dry in the form of hard toast or baked bread with or without a little salt butter. To be properly baked the bread should be cut into slices and put into an oven that is hot enough to make it, through and through, of a yellow biscuit colour in about an hour's time. Prepared in this way it remains crisp and palatable for a long time. The meal of boiled bread and milk, sweetened with sugar, that is often given is an ideally bad food for little children, and is almost certain to be gulped down without mastication or insalivation. When the food is given dry it has much more taste, and this promotes its mastication and also increases the secretion of the gastric juice. It also excites a free flow of saliva, which leads to the starchy particles about the teeth being dissolved and washed down. Even before the appearance of the molars a little food of this kind may often be given with advantage, though it is, of course, only when they are through the gum that thorough mastication is possible.

The other important point is that the infant's food should not be unnaturally sweetened. Children fed on tasteless pap usually have a craving for cane-sugar, and it is a common error to suppose that it is therefore a necessary and natural thing to give them. This is far from being the fact; indeed, the case against sugar being used in diet as freely as it is at present is very strong. Cane-sugar, we must remember, is a comparatively recent addition to ordinary food in the world's history. In old days it was quite unknown; even in Pepys' time it was still an expensive luxury. A few hundred years ago the children of our own and other races, whose teeth are known to have been better than ours are now, were brought up altogether without it. Again, while it is true that sugar is an important element in some of the best natural foods we have—such as fresh fruit—it is scarcely anywhere found in Nature (except in honey) without being in combination with acid salts, and so mixed with fibrous tissue that the mouth and jaws are well exercised in the process of obtaining it. To give it freely in the form of jam, chocolate,

and sweets is obviously unnatural. When given in these forms, in any quantity, it interferes with the proper amount and composition of the saliva, lessens the child's power of digesting starchy food, and spoils his appetite for the fatty foods which are so important for his growth and development.

We may sum up, then, by saying that although sugar is a welcome addition to children's diet, and, in moderation and with sensible safeguards, harmless and useful as well as pleasant, its use must be limited and regulated by common-sense. Given carelessly, and in huge excess, as it often is in this country, it is a fruitful source not only of dental caries but also of serious indigestion.

Not only must the child's meals contain food in proper forms, but they should also be arranged as far as possible physiologically. That is to say, they should not end with sticky carbohydrates which are likely to lodge about the teeth, or with sweets. The dentists tell us that the ideal thing is that milk puddings, bread and jam, and all sugary articles of diet should if possible be followed by some form of fresh fruit, and that the best of all fruits for the teeth are ripe apples. Their slight acidity promotes a free flow of saliva of high alkalinity and diastatic power, and in chewing them the teeth are naturally and efficiently scoured. When the meal cannot be ended with fruit, which must very often happen, it is practically sufficient if the mouth is well rinsed out with water, and it is even better if a mouth-wash of an acid reaction (Appendix E, Form. 33 and 34) is used to promote a free secretion of alkaline saliva.

The common habit of giving a child chocolate or other sweets at the end of any meal is altogether bad, unless the mouth is washed out after. The worst thing that can be done in this way, however, is to encourage the fermentation in the mouth by giving the child a sweet after supper when he is going to sleep; a "tiny piece of chocolate" is as harmful in this respect as a bagful of sweets.

The subject of the child's diet is further dealt with in Chapter VIII.

Artificial Methods of Cleansing the Teeth.

The Tooth-Brush.—The real use of the tooth-brush is to keep the teeth white and to remove discoloration. It is less necessary and also less efficacious for this purpose in children

than in adults. It is very difficult to use a tooth-brush so as to be an efficient preventive of caries; for, however carefully it is manipulated, it never removes all the fermenting matter from the teeth, so that it does not really cleanse them as efficiently as rinsing out the mouth or as the vigorous chewing of suitable food.

The tooth-brush must be as small as possible, with fairly stiff bristles, and it should not be used too vigorously for fear of injuring the margins of the gums. When not being used, it should be kept in a weak antiseptic solution, or else it should be frequently washed with soap and water. Plain water, and no dentrifice, should be used with it. Before and after its use it is a good plan to rinse the mouth out with an acidulated mouth-wash.

Tooth-Powders.—Like tooth-brushes, tooth-powders are more useful for cosmetic purposes than for preventing decay. If used at all, they must not be too gritty in consistence or they may damage the enamel. If they are alkaline they neutralise the acids of fermentation for the time being; but they also discourage the secretion of the saliva and lower its cleansing properties, so that their use is really inadvisable.

Antiseptics.—When the part played by bacteria in the production of caries was first demonstrated by Miller, much was expected from the use of antiseptics. In practice, however, their effect has proved disappointing. The micro-organisms are far too safely embedded in the carbohydrate debris to be freely reached by them, unless the antiseptics are used in unpleasantly strong solutions and retained for an impracticably long time in the mouth. They also all tend to retard the liquefaction of albuminous debris about the teeth. When a wound has been inflicted, as by the extraction of a tooth, the case is different, and an antiseptic mouth-wash is beneficial.

Mouth-Washes.—The simplest and most natural plan for keeping the mouth clean is to rinse it out with plain water after each meal. The use of this simple proceeding is most efficacious in preventing caries; and, if it is done regularly and thoroughly, it is probably all that is necessary to keep the teeth healthy, if they are normal in form and not overcrowded. Dentists, however, tell us that it is best to use, instead of plain water, a mouth-wash containing a vegetable acid salt (such as acid tartrate of potash) and an aromatic flavouring agent. Such mouth-washes (Appendix E, Form. 33 and 34) strongly stimulate

the secretion of normal saliva and thus promote the natural cleansing of the teeth.

The best and only thoroughly satisfactory method of keeping the teeth clean and preventing caries is therefore the natural and physiological one of the vigorous chewing of suitable food. And this can be usefully supplemented by the regular washing-out of the mouth, morning and evening and after each meal, with water or with a suitable mouth-wash. It is especially important that this should be done thoroughly before the child goes to bed, as it is during the night that the bacterial growth is most active. It is said that, when reasonable habits of mastication are practised, it takes twenty-four hours for the mucus and debris to reaccumulate to a harmful extent round the teeth.

There are some medicines the taking of which is bad for the teeth—chiefly those containing a mineral acid, and some preparations of iron. Taking them through a glass tube does not protect the teeth efficiently; and the best thing to do is to see that the child washes the mouth out freely each time after the medicine is taken.

Toothache.—The occurrence of toothache due to dental caries is evidence of neglect; for, by the time the pain begins, the decay is often so far advanced that the tooth is past saving. It is most important for its prevention that the disease should be dealt with early. For this reason a careful watch should be kept for its beginning, and *every child from the second year should have his teeth examined by a dentist twice a year*. Caries is much more rapid and serious in its effects in temporary than in permanent teeth, and requires early and energetic treatment because of the rapidity with which it sets up osteomyelitis of the tooth and necrosis of the pulp, with abscess at the root. This is due to the pulp being relatively larger than in adults, and also separated from the enamel by a much smaller thickness of dentine.

The treatment of toothache is, of course, a matter for the dentist; but a few simple measures may be mentioned which are sometimes useful in relieving the pain until more skilled treatment can be obtained. One of the first of these that should be tried consists of filling the mouth with a hot solution of bicarbonate of soda; this often affords considerable relief in the pain of dental caries. When the pain comes from a commencing

gumboil it may be benefited by irrigation with hot saline solution; but moist heat should never be applied to the outside of the jaw in such cases. When the pain originates in an accessible cavity, a fragment of cotton-wool dipped in pure phenol or clove oil and inserted into it often stops the aching for the time. A dose of antipyrine, aspirin, or other similar analgesic is sometimes useful in dulling the sensation, and a dose of Gregory's powder or a saline purge is also often followed by relief.

Dental Abnormalities.

Most of the abnormalities of form of the teeth, and of their number and position, are mainly of interest to the dental surgeon and pathologist. There are, however, certain changes in their form which have a wider significance as giving information relating to the general health or past history of the child. Sometimes there occurs a hereditary and familial absence of certain teeth.¹

Traces of Grinding the Teeth.—We often find indications that a child grinds his teeth during sleep. It is shown by extreme flattening of the tips of those teeth which are most prominent, as if a file had been used upon them; and the canines are those most strikingly affected. This appearance may arise in children to a slight extent from mere chewing; it is only seen in a severe degree when there has been habitual grinding of the teeth for a long time. It may therefore be looked upon as a physical sign of nervous irritation. The irritation which it indicates is generally of the comparatively harmless sort which results reflexly from injudicious feeding, or the presence of worms, or from too much excitement in a nervous but otherwise healthy child. If we are told that a child who has severely ground teeth sleeps quietly and well, we may suspect, as Dr Warner says, "that it is the nurse who sleeps soundly, and not the child."

Defects in the Calcification of the Enamel.—These are often seen in the permanent and more rarely in the temporary teeth, forming a discoloured transverse furrow which passes across the front of the incisors and canines, and often also implicates the cusps of the molars. This, as we have already seen, shows that at the period of the child's life at which the

¹ M. E. Rutherford, *Brit. Med. Journ.*, 1931, ii., 9.

calcification of that part of the enamel was proceeding, something occurred to check its progress. The condition is analogous to the grooving of the finger-nails which we observe after fevers. A groove across the middle of the central permanent incisors may be due to an acute illness which took place in the third or fourth year. Many grooves of this kind, however, occur, for which no corresponding history of illness can be obtained.

Mercurial Teeth.—This is the name given by Sir Jonathan Hutchinson¹ to an abnormality of the permanent teeth due to interference with the development of the enamel. In his opinion it may arise from simple stomatitis, as well as from

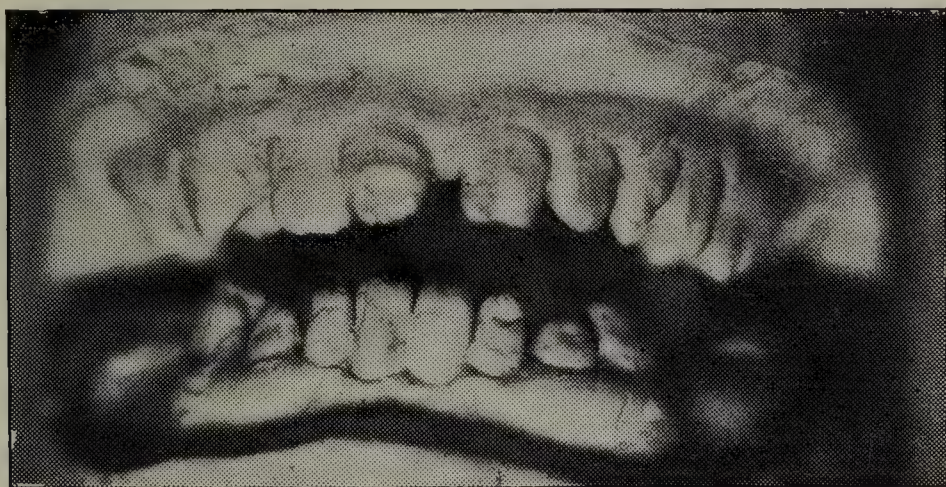


FIG. 27.—Congenital syphilitic teeth : upper central incisors.
(Girl of 9 years.) (Dr R. J. Mackessack's case.)

that which follows the administration of mercury. The teeth are rugged, pitted, or dirty on the surface. The first molars, incisors, and canines are the teeth which show the condition most frequently; the premolars escape. The effect of the stomatitis is usually confined to an interference with the development of the enamel, and rarely of the dentine. It does not cause arrest of development of the tooth as a whole in the way that congenital syphilis does.

Congenital Syphilitic or Hutchinson's Teeth.—(Figs. 27 to 29). This variety of dental abnormality is important, because, as Hutchinson says, "It is, if taken alone, by far the most valuable of the signs by which we recognise in adolescents the effect of inherited syphilis." The characteristics of these teeth are not sufficiently known, and abnormal and peculiar teeth of other kinds are often erroneously regarded as proofs

¹ *Illustrations of Clinical Surgery*, 1878, i., 53.

of congenital syphilis. The main points about "Hutchinson's Teeth" are as follows:—

1. It is always *the permanent teeth* which are affected thus. The temporary teeth in syphilitic infants present no special peculiarities of form.

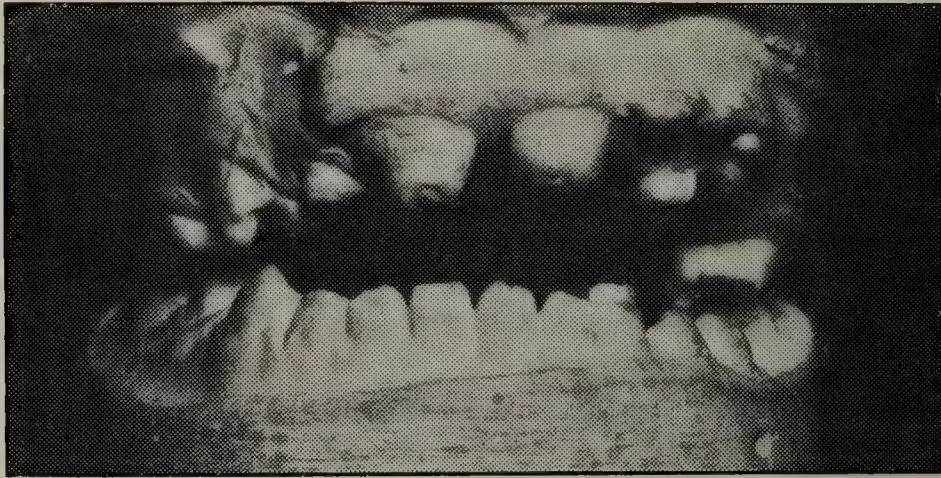


FIG. 28.—Congenital syphilitic teeth: upper central incisors. (Boy of 10 years.)



FIG. 29.—Congenital syphilitic teeth: upper jaw. Showing pegged and rounded central incisors and domed molars. (Girl of 10 years.) (Dr R. J. Mackessack's case.)

2. The only teeth which afford incontestable evidence of congenital syphilis are the *upper central incisors*. The lower central, and less seldom the lateral, may supply corroborative evidence, but they cannot be trusted to alone.

3. The characteristic peculiarities which distinguish these central incisors are as follows: They are *dwarfed*, being too short and too narrow; and sometimes the portion of the upper jaw from which they grow is also arrested in growth. They often *stand somewhat apart* and slope towards one another. They are unusually *rounded* on section; they are "*pegged*" (that is to say, the teeth are broader near the gum than at their free edge); and they are *notched*. The notch is usually shallow and the dentine is exposed at the bottom of it. It is formed by the breaking away of the imperfectly developed central portion of the edge. The teeth are generally *not of a good colour*, and they are so *soft* that, by the time the patient is twenty, they are often ground down like those of an old man.

The first molars also suffer from syphilis and may show the characteristic dome-shaped crown due to arrest of growth of the central denticles (Fig. 29). Their sides slope towards the centre, over which the enamel is defective. These teeth, however, show marked changes due to the administration of mercury and the two types of malformation become so inextricably mixed that, according to Hutchinson,¹ they are very unreliable indices of the syphilitic infection.

¹ J. Hutchinson, *Syphilis*, London, 1889. Commentary ccvii.

CHAPTER IV

EXAMINATION OF THE HEAD, NECK, BACK, AND LIMBS

The Head

THE size, shape, and ossification of the cranium, including the condition of the fontanelle and sutures, are the main points to which attention should be directed in examining the head.

Size.—At birth, the circumference of the infant's head is on an average 13 to $13\frac{3}{4}$ inches; and it grows so rapidly that



FIG. 30.—Microcephalus in girl
of $4\frac{3}{4}$ months.

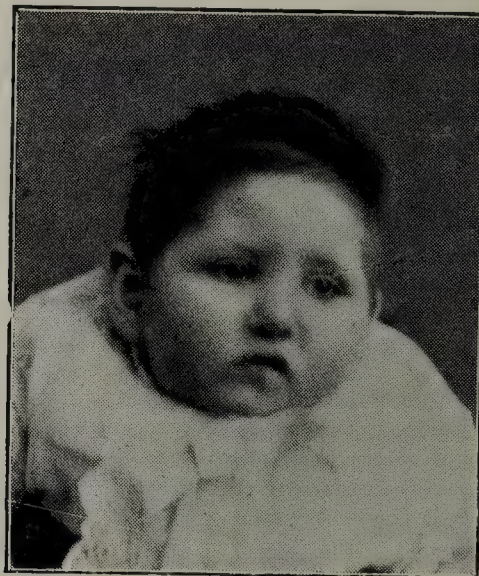


FIG. 31.—Microcephalus in
girl of 2 years.

it usually gains 3 inches in the first six months, and by the end of the first year measures about 18 inches. During the second year the circumference increases by about 1 inch. At five years the average measurement of the child's head is from 20 to $20\frac{1}{2}$, and at ten about 21 inches.

The size of the head may vary considerably within normal limits, and sometimes children are seen with curiously long or large heads, whose intellectual development is not abnormal. As a general rule, where the head is unusually small or large from causes which interfere with the health of the brain, some



FIGS. 32 and 33.—Hydrocephalus in girl of 4 months.



FIGS. 34 and 35.—Hydrocephalus in boy of 18 months.



FIGS. 36 and 37.—Enlarged head from old Hydrocephalus in boy of 12 years.

characteristic change in its shape will also be found. It is only when the variations in size are great that they are to be considered really morbid. In a large proportion of mentally defective children the head is below the average in size, and this is an important contributory point in the diagnosis and prognosis.

Shape.—Asymmetry of the cranium is frequently seen in infants who are otherwise normal or merely rickety, and the condition is of no practical importance. The head may be lop-sided—prominent on one side in front and on the other side behind. Occasionally a similar deformity of the chest accompanies the asymmetry of the head.

Microcephalic crania (Figs. 30 and 31) are recognised not only by their small size, but also by their peculiar shape.



FIG. 38.—Square Head of Rickets. (Boy of $2\frac{1}{2}$ years.)

The forehead is very narrow and receding, with a medial ridge, the vertex somewhat pointed, and the occiput flat. The fontanelle is either closed at birth or closes prematurely.

In *hydrocephalus* (Figs. 32 to 37) the head is enlarged both antero-posteriorly and from side to side, and its shape is more globular than in any other condition. In early advancing cases we are often helped in the diagnosis by observing a downward direction of the eyes, which is shown by the high level at which the lower lid crosses the eyeball and the unusual amount of sclerotic seen above the iris. The large and bulging fontanelle, which is often patent up to four or five years of age, is also a striking feature, as well as the thinning and defective ossification of the cranial bones resulting from internal pressure. When the head has recently enlarged, the superficial veins of the scalp are usually dilated. A mild degree of hydrocephalus, as is frequently present in cerebral tumour, can often be detected

by the presence of a cracked-pot sound on percussion due to separation of the sutures (Fig. 232, p. 749).



FIG. 39.—Square Head of Rickets.
(Boy of 3 years.)

The chief characteristic of the ordinary *rickety cranium* is its squareness (Figs. 38 and 39), the top of the head, the sides, and the face all tending to be flatter than usual. The head itself is usually rather larger than it should be for the child's age, and a good deal too large for the size of his body.

In many cases of rickets the form of the head becomes further changed by the outgrowth on its surface of osteophytic nodes, or bosses, as they are usually called. These localised thickenings on the skull sometimes grow until the bone is fully half an inch in thickness, and are situated over the frontal and parietal eminences (Fig. 40).

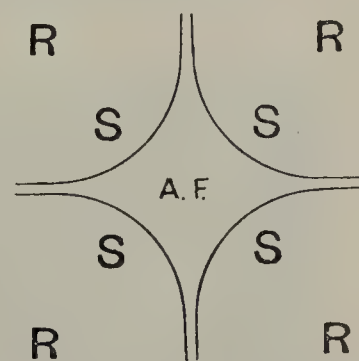


FIG. 40.—Diagram showing relative positions of Syphilitic Nodes (S) and Rachitic Nodes (R) with reference to the Anterior Fontanelle (A.F.).

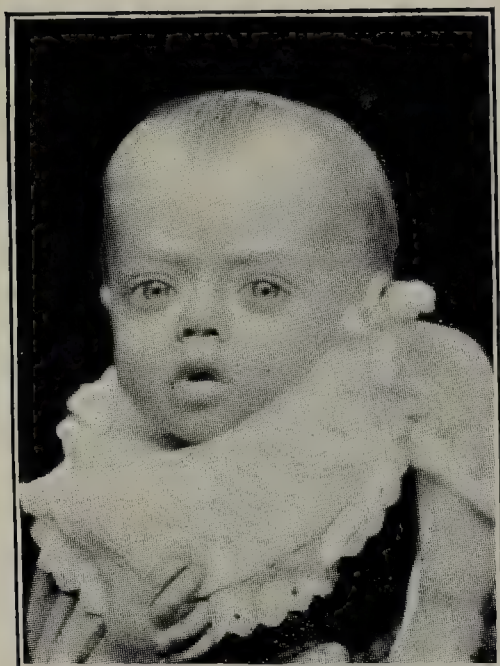


FIG. 41.—J. S., at 12 months.

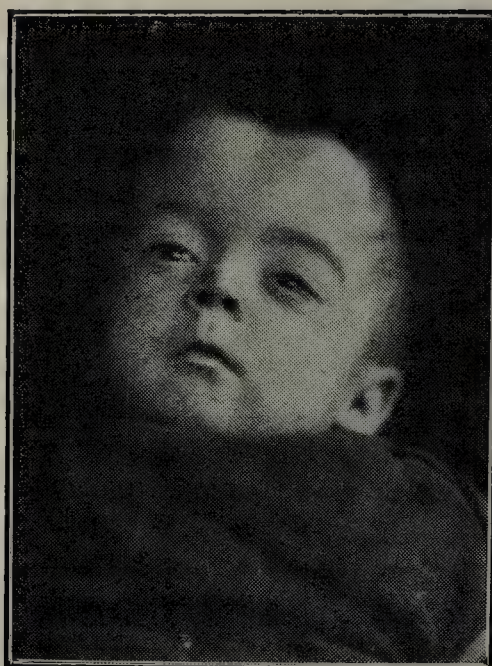


FIG. 44.—J. P., at 12 months.

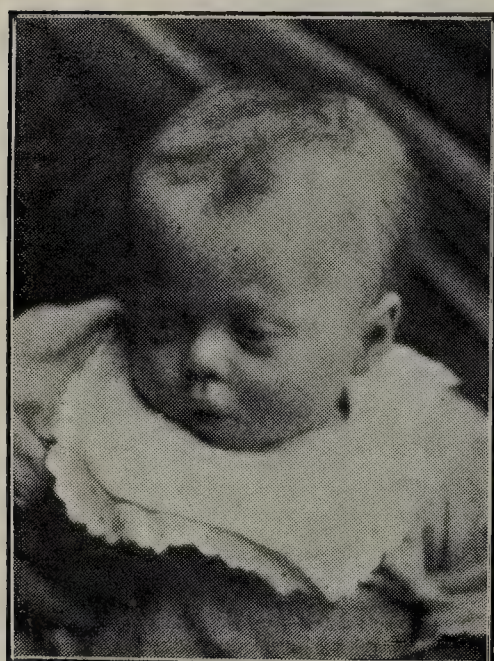


FIG. 42.—J. S., at 2 years.



FIG. 45.—J. P., at 2 years.

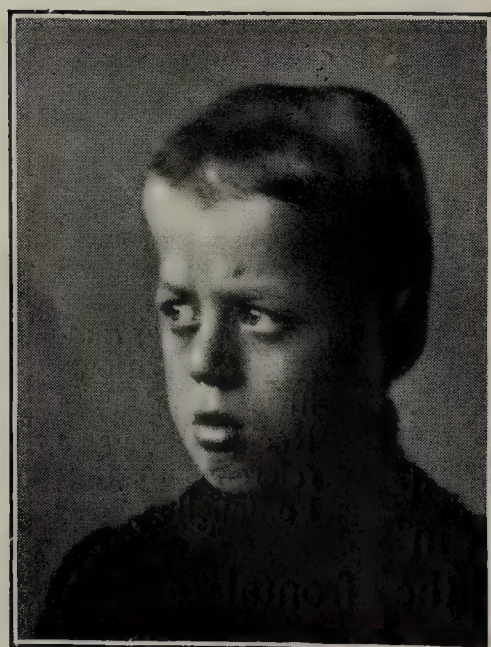


FIG. 43.—J. S., at 11 years.



FIG. 46.—J. P., at 11 years.

In *syphilis* also the head assumes a definitely square shape and may simulate that met with in rickets, but the osteophytic nodes or so-called bosses develop more around the anterior fontanelle (Fig. 40). When these bosses are unduly prominent there are grooves between them, which suggest the hot-cross-bun or nates, and hence the descriptive terms "hot-cross-bun" or "natiform" skull. Figs. 41 to 46 illustrate the appearances in this type of bossing.



FIG. 47.—Tuberculous Disease of the Frontal Bone.

Peculiar and characteristic deformities of the cranium are found in osteogenesis imperfecta (p. 880), in the variety of fragilitas ossium which is associated with blue sclerotics and otosclerosis (p. 882), and in oxycephaly (p. 884).

Soft, often asymmetrical, swellings on the surface of the cranial bones are sometimes formed by abscesses over *tuberculous bone disease* (Fig. 47); similar swellings may, less commonly, be met with in congenital syphilis, and, rarely, in sarcoma of the suprarenal gland (p. 521) and chloroma (p. 485).

Irregular hard outgrowths are often felt on the surface of the cranium in cases in which there has been a cephalhæmatoma (Fig. 48) which has been long in being absorbed. They are due to bone having been formed on the under surface of the

pericranium. They are of no consequence and disappear spontaneously in course of time.

The Ossification.—This is investigated by palpating the anterior fontanelle, the sutures, and the parietal and occipital regions for craniotabes.

The Fontanelle.¹—In the cranium of the new-born child there are six fontanelles situated at the various corners of the parietal bones. Except in some cases of rickets and hydrocephalus, the posterior and the four lateral fontanelles are usually closed within a few weeks of birth. The anterior,



FIG. 48.—Double Cephalhæmatoma.

therefore, is the only one which is of much clinical importance and it is generally spoken of as “the fontanelle.” The characteristics of the normal fontanelle which have to be noticed are its shape, edges, tension, level, pulsation, and size.

In *shape* the fontanelle is somewhat rhomboid with slightly curved borders which are convex inwards. Its bony *edges* are felt distinctly at their junction with the membrane, and are somewhat rounded. Should they be so thin and yielding that there is difficulty in determining where the bone ends and the membrane begins, this indicates cranial rickets or long-continued increase of the intra-cranial pressure, or both.

The fontanelle, like the eyeball, has a normal *tension*. The membrane is stretched somewhat tightly between the bony

¹ See C. Hochsinger, “Studien über die klinischen Verhältnisse der Stirnfontanelle,” *Wiener Klinik*, July, August 1892.

edges, and is about the same level as they are. Any change in the intra-cranial pressure affects the tension and *level* of the fontanelle, so that it either bulges out above that of the surrounding bones or becomes depressed and hollowed out. There is normally a rhythmic *pulsation* of the fontanelle transmitted from the intra-cranial arteries. When investigated by the sphygmograph,¹ the tracing of this pulsation is found to have an anacrotic character, and it also shows slight irregularities due to the respiratory movements (Fig. 49). The pulsation is increased if the tension is moderately raised, but ceases if it is either greatly raised or lowered to any considerable extent.

The *size* of the fontanelle is important. It is usually stated that the fontanelle closes during the second year—between the

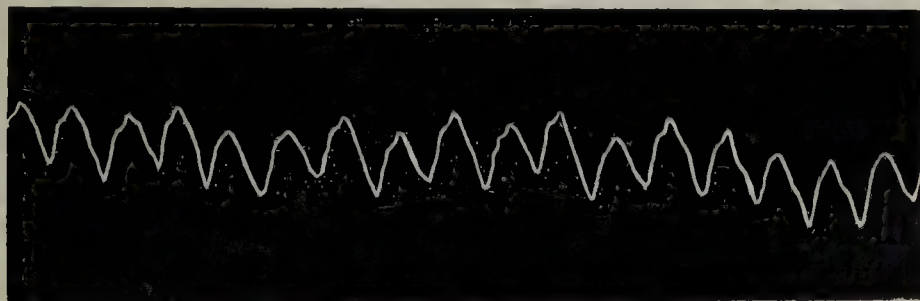


FIG. 49.—Tracing of normal fontanelle pulse from an infant of 4 months. Dudgeon's sphygmograph. (Dr H. O. Nicholson.)

fifteenth and eighteenth months—but this is due to the fact that most observations on the question have been made in the hospital class of patient, who is of course prone to rickets, which is the commonest cause of delay in closure. In the better-class child or where rickets can be eliminated, the fontanelle is usually closed during the first year and may be quite imperceptible even at nine months.

The conditions of the fontanelle which are most important from a clinical point of view are as follows :—

I. *Alterations in size and date of closure.*

(a) An abnormally large size of the fontanelle, or abnormal delay in its closure, may be due to various causes. It may arise from hydrocephalus or any other morbid condition which increases the cranial contents. Much more commonly it is caused by rickets—so that if the fontanelle is large at eighteen months, or open at all after the second year, rickets should

¹ H. O. Nicholson, art. "Pulse," *Encyclopædia Medica*, x., 208 : R. Hutchison and George Elder, *Edin. Hosp. Rep.*, 1895, iii., 268.

always be suspected. Delay in closure is also common in mongolism, while in untreated cretins and in cases of cleidocranial dysostosis the fontanelle generally shows no tendency to close in infancy, and may be found widely open even in adults of thirty or forty years old. Simple malnutrition does not seem to delay its closing.

(*b*) Premature closure is a valuable early sign of microcephalus, and occurs to a less extent in many cases of birth-injury to the brain.

2. *Alterations in the tension and level of the fontanelle.*—These are extremely important owing to the information they supply as to the intra-cranial tension ; thus—

(*a*) Slight increase of its tension with some bulging outwards is caused by cerebral hyperæmia, active or passive. We see it therefore in acute feverish conditions, in whooping-cough or chronic bronchitis, and always, temporarily, when the child coughs or cries.

(*b*) Great tension with marked bulging and absence of pulsation indicates a considerable increase in the contents of the skull, such as is found in effusion into the ventricles or pia-arachnoid in meningitis. It is seen also in new-born children when there has been an extensive intra-cranial hæmorrhage.

(*c*) Abnormal depression with diminished tension of the membrane is associated with lowering of the vital powers. It is met with in acute cases of diarrhœa, with rapid loss of fluid. It is very characteristic of cases of atrophy (marasmus), of food-intoxication (Chapter IX.), and of wasting from any cause. Recovery of the normal tension in such cases is a reassuring sign of improvement.

In cases of diarrhœa with cerebral symptoms ("hydrocephaloid") the presence of this sign is useful in helping to distinguish the condition from real hydrocephalus (meningitis). When it is present to a marked degree in diarrhœa it is an urgent indication for stopping milk, administering saline infusions, and applying external warmth.

(*d*) A condition of normal tension of the fontanelle, along with cerebral or meningeal symptoms, is often met with and is of diagnostic importance. It occurs, for example, in some cases of "cerebral" pneumonia (that is, cases of pneumonia with prominent cerebral symptoms, such as delirium, head-retraction, and fits), in pyelo-nephritis and enteric fever, and its presence is

sometimes of value in differentiating these from cases complicated by meningitis. Generally speaking, it may be said that in feverish illnesses in young babies cerebral symptoms do not indicate the presence of intra-cranial disease unless they are accompanied by a bulging fontanelle, but as the elimination of meningitis is not possible on this ground alone lumbar puncture should always be performed when there is the least suspicion of meningeal mischief.

The Sutures.—The main sutures should also be felt. Any gaping of them with thinning of their bony margins has the same significance as enlargement of the fontanelle.

Craniotabes.—Craniotabes is the name given to thinning of the cranial bones. In its slightest form it causes a softening of their edges, with widening of the fontanelles and sutures, such as has been already referred to. When it is present to a severe degree we find little rounded areas of thinned bone at some distance from the sutures. These yield before the point of the finger, and give a slight sensation of crackling like that of parchment. They are found most frequently on the parietal and occipital bones near the lambdoidal suture, and sometimes the absorption of bone goes so far that there is nothing but a little window of membrane left at each of the affected areas.

In osteogenesis imperfecta and some other obscure conditions, considerable areas of the cranial bones are membranous at birth, and remain so for months. Craniotabes is sometimes due to chronic hydrocephalus. In the majority of cases it is a manifestation of rickets. Craniotabes is present to some degree in most young babies in whom that disease is well marked, but it is usually observed only in the very young infant and at a stage when no other clinical evidence is present.

Cephalic Bruits.—If the ear or the end of a stethoscope is applied over the open fontanelle, or in older children over the external ear, a blowing systolic murmur can often be heard. The occurrence of this murmur has long been known, and it has generally been recognised that it has no diagnostic significance. As it may sometimes, however, be a source of anxiety to parents, it is, as Still¹ points out, not altogether devoid of clinical interest.

¹ *Brit. Journ. Child. Dis.*, Oct. to Dec. 1921, xviii., 173. See also Henoch, *Beitr. z. Kinderheilk.*, 1861, 170, and Osler, *Boston Med. and Surg. Journ.*, 1880, ciii., 29.

In older cases the murmur may be heard by the child himself when his ear is closed by his hand or the pillow, and his mother may hear it when she puts her ear against his. The medical man can hear it best by using a binaural stethoscope, the chest-piece of which has been replaced by the head-end of another binaural stethoscope, the ear-pieces of which are inserted into the child's ears. What is heard is a blowing systolic murmur clearly of arterial origin, and quite unlike the continuous hum of a venous bruit. Still suggests that the tortuosity of the carotids at the base of the cranium may have something to do with its causation, and that their straightening out in the course of development may account for the usual disappearance of the murmur in later life. Neither rickets nor anæmia is necessary for its production.

The Neck.

In examining the neck we have to look out for any restriction of its natural movements, for tumours in the sternomastoid, if the patient is a young baby, and for enlargement of the lymphatic glands.

Rigidity of the Neck in babies and its **retraction** represent two degrees of a common and important symptom which will be referred to later in considering the diagnosis of nervous disease (Chapter XXVII.).

In older children it occurs in an acute and temporary form as a manifestation of "lithæmia or rheumatism," and as a permanent deformity in the wryneck which often follows sternomastoid tumour, in rheumatoid arthritis, and in cervical caries. A sudden very painful rigidity of the neck is sometimes caused in cases of septic sore throat by acute inflammation of the deep lymphatic glands which lie in close contact with the cervical vertebræ.

Enlargement of the Cervical Lymphatic Glands is always a matter of interest, and it is important to notice which group of glands is affected. The area of skin or mucous membrane with which these are connected should always be examined, so as to discover, if possible, whether there is any local source of irritation present which requires treatment.

Enlargement of the submaxillary group of glands, if there is no visible cause for it on the face, generally indicates some

irritation from the gums or teeth; and in the same way sores on the chin, the lower lip, or the front of the tongue give rise to enlargement of the suprahyoid gland. If a number of superficial enlarged glands are found along the posterior border of the sternomastoid muscle, there probably is, or has recently been, some irritation in connection with the scalp, such as that produced by pediculi. Swelling of the posterior cervical glands occurs in pharyngitis and is also often the first symptom of rubella; and it may sometimes be seen in cases of erysipelas of the scalp, before the rash appears.

The upper set of deep cervical glands near the angle of the jaw drain the naso-pharynx, pharynx, and neighbouring parts; and, as their area of distribution includes a very large amount of adenoid tissue, they are more frequently affected than any others. That one situated at the angle of the jaw is often spoken of as the tonsillar gland because it is so frequently affected in disease of the tonsil. Their rapid enlargement on one or both sides generally signifies some acute infection in the area which they drain; and conversely, if they are normal it is in favour of there being no serious or acute throat affection. When they are chronically enlarged the cause is generally tuberculosis.

In the normal individual no superficial glands should be palpable except in the groin. Enlargement of the glands in any other situation is abnormal, although in childhood, because of the relatively increased activity of the adenoid tissue, a slight degree of enlargement has not the significance that it has in the adult. In the hospital class of child, owing to the frequency of infection of the upper respiratory tract and of the skin, a moderate degree of enlargement of cervical and axillary glands is almost constant. Enlargement of the thoracic glands is frequently, but in our opinion erroneously, considered indicative of intra-thoracic tuberculosis. Similarly, the enlargement of the cubital or epi-trochlear glands cannot be considered pathognomonic of syphilis.

The Back.

In the healthy child the spine is characterised by great suppleness in all its varied movements. Even a small degree of *stiffness*, therefore, and the least *pain on normal movements* are to be regarded as important morbid signs; they generally

indicate commencing spinal caries. Also, whenever a persistent or recurrent pain in the abdomen or lower limbs is complained of, it should always direct attention to the spine.

It must be remembered that in the infant, and even in the young child while sitting, the back assumes a kyphotic appearance which is often mistaken by the parents for a pathological curvature. The same mobility causes the child's spine when in the erect posture to incline to what would in the adult be called lordosis.

Rickety curvature of

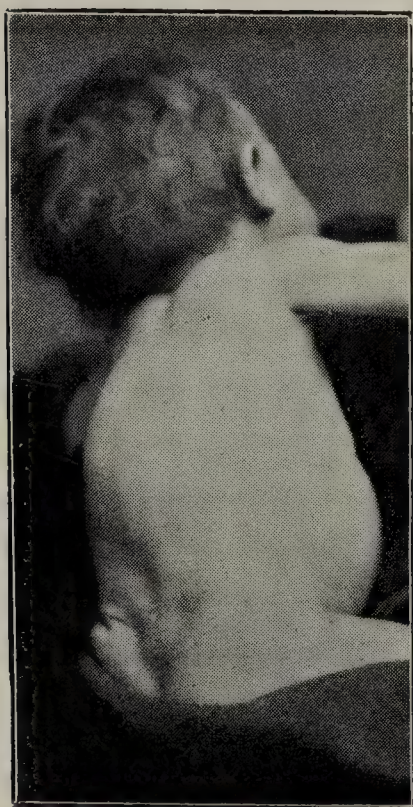


FIG. 50.—Rickety Curvature of Spine in boy of 20 months.

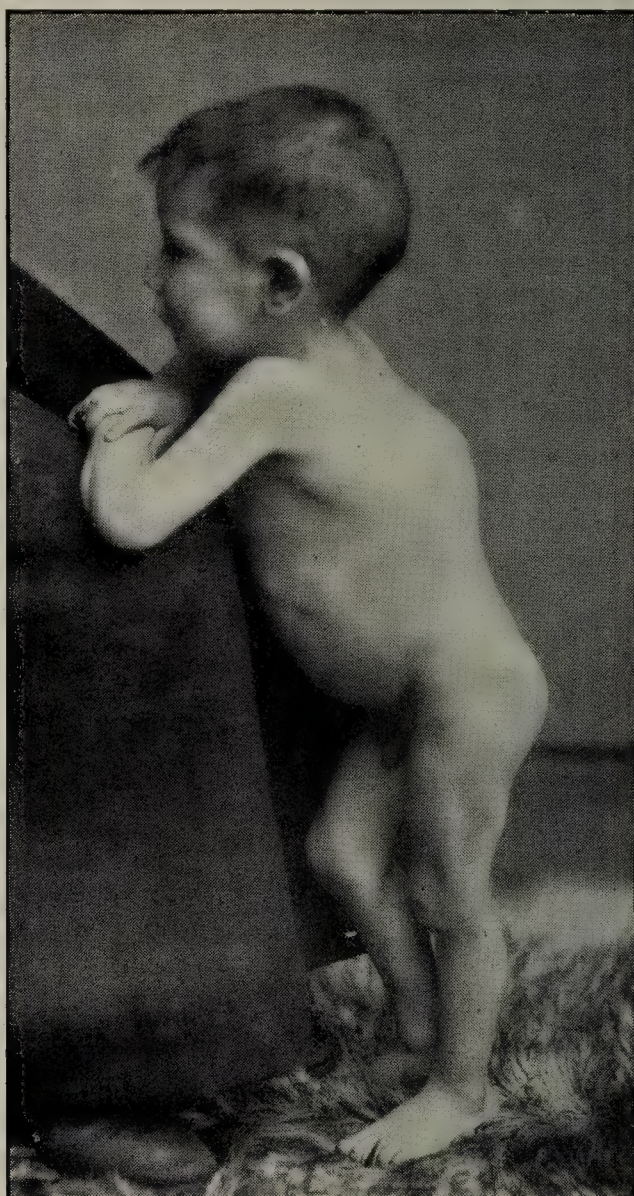


FIG. 51.—Spinal Caries, showing acute curvature and characteristic attitude.

the spine is a very common condition (Fig. 50). In most cases it takes the form of kyphosis, and the curvature differs from that of Pott's disease (Fig. 51): *first*, in forming a wide curve and not an acute angle; *secondly*, in disappearing when traction is made on the legs, except in severe and long-standing cases; and *thirdly*, in being unaccompanied by pain on passive movement, or by distinct stiffness.

Paralysis of the muscles of the back is sometimes diagnosed as spine disease. We have known this mistake made in cases

of diphtheritic paralysis, in paralysis due to tumour of the cord, and in poliomyelitis.

Lordosis, resulting from congenital dislocation of both hips, is sometimes mistaken for spinal disease. The parents of mentally defective children often attribute their inability to sit up to disease of the spine.

Compensatory twisting of the spinal column such as is caused by a short leg, by wryneck, or by paralysis of the spinal accessory nerve is apt to be mistaken for ordinary lateral curvature.

The Limbs.

In examining the limbs, the colour and temperature of the skin are to be noted. The size and consistence of the muscles, the length and outline of the bones, and the presence of any tenderness or pain in these structures are also to be observed. The joints and the hands and feet call for special attention.

The Joints.

In the examination of diseased joints the following points are of importance:—

1. The *child's age*.—Some diseases only occur in infants; others only in older children.

2. The *nature of the onset* of the disease.—Whether it began insidiously or suddenly, whether it followed an illness or injury, and whether it was accompanied by any constitutional symptoms such as fever, anæmia, or wasting.

3. The *distribution* of the lesion.—If one or several joints are affected, and whether the lesions are symmetrical. We have also to search for signs of disease elsewhere which may throw light on the joint condition—for example, any affections of the bones or of the tendon sheaths, abscesses anywhere, disease of the heart, lungs, abdomen, lymphatic glands, teeth, eyes, or other parts.

4. The *local conditions* to be investigated by inspection, palpation, and questioning include:—

(a) The *posture* of the limb—if kept flexed or extended.

(b) The presence of *muscular wasting*.

(c) The *swelling* of the joint—its exact position and characters. This may throw light on the extent to which the

bones, ligaments, and synovial membranes are severally implicated.

(*d*) *Redness* of the skin or *distension of its superficial veins*.

(*e*) *Local heat*.

(*f*) *Fluctuation* ; or *thickening of the synovial membrane*.

(*g*) *Mobility*—if lessened or increased in freedom and extent.

(*h*) The presence of *crackling* sensations on moving the joint, or of any creaking of the tendons or bones.

(*i*) *Tenderness* on being touched, or pain on passive or active movement.

(*j*) Any history of *spontaneous or other pain*, either constant or intermittent—as in the case of the “lightning pains” of tuberculous hip disease.

5. An *X-ray examination* of the affected joint should, if possible, be made in *all* doubtful cases, as it often throws valuable light on the diagnosis.

The following are the most important joint diseases met with in children:—

Traumatic Synovitis.—Joint affections due to injury are, of course, far less common in little children than in schoolboys or young men. It should be remembered that a history of an injury is often given in cases which turn out to be tuberculous, syphilitic, or rheumatic. The onset in a case of traumatic synovitis is usually sudden, and there is an absence of disease elsewhere. Should the joint have been severely injured, there will generally be evidence of bruising visible under the skin within a day or two. The pain will usually be severe and the tenderness great.

We have several times seen a mild form of traumatic synovitis of the hip-joint in young children (three to six) due to sudden forcible abduction of the limb. In some cases it was caused by the child slipping on a polished floor with his legs widely separated. In others it followed a too vigorous attempt to rival the gymnastic feats of a bigger boy. Rapid recovery followed rest in bed.

While speaking of injuries it may be mentioned that in children the *elbow-joint* is much more frequently hurt than any of the other articulations. Both dislocations and fractures are common in this region, as well as bruising and separation of the epiphyses.

An injury to the arm, which is not uncommon in young

children, is what is called *Pulled Elbow*. This condition is apt to be mistaken for traumatic synovitis. It consists in a subluxation of the head of the radius out of the orbicular ligament. The usual cause of the injury is a sudden jerk given to the arm when in the position of extension. It is apt to occur when a nurse tries quickly to stop a young child, who has tripped, from falling. Immediately after the injury the arm hangs down, as if paralysed, and is pronated and slightly flexed. Pressure over the head of the radius and all movements of the elbow cause sharp pain. Voluntary supination is impossible. The prognosis as to complete recovery of movement is very good, but the accident is apt to recur. The treatment consists in forcibly extending and supinating the forearm, while at the same time pressing the head of the radius back into its place. The arm is then fully flexed, and put up for two or three days in a sling.

Tuberculous Joint Disease.—Tuberculosis is the commonest cause of disease of the joints in children, and it may affect them at any age (Chapter XXXVII.). Its onset is often insidious but sudden exacerbations from rupture of a focus in the bone into the joint are not uncommon. A distinct history of recent injury is frequently given; and there have often been fever, anæmia, and wasting before the joint affection was noticed. The joint disease is sometimes single, sometimes multiple, but usually not symmetrical. Other tuberculous lesions may be present in the bones, glands, lungs, abdomen, or elsewhere.

The affected limb is kept flexed, and there is generally well-marked muscular wasting. There may be little or no redness, but the local temperature is usually slightly raised. The amount and character of the swelling vary, but whenever the affected joint is at all superficial in its position the synovial membrane will be found to be thickened. Slight rigidity, with limitation of movement, is an early sign. If the affected joint is in the lower limb, lameness is generally the first symptom noticed. The character of the pain varies greatly in different cases. Sometimes it is severe. This is often so in hip-joint disease, in which it is felt in the neighbourhood of the knee. "Starting" or "jerking pains" at night are characteristic of hip-joint disease. In many of the other joints, however, pain is often almost entirely absent in early tuberculous disease.

Arthritis from Congenital Syphilis.—This condition is rare in babies. It is generally met with about the time of the second dentition (five to fifteen). The onset is usually insidious and there is little or no constitutional disturbance, the child seeming merely a little out of sorts. Clinically there are four types of syphilitic arthritis—(a) the painless *hydrops articuli*; (b) the acute syphilitic arthritis; (c) arthritis in association with syphilitic osteitis, and (d) pseudo-rheumatoid arthritis.

The most characteristic variety is the painless hydrops, which usually involves both knees, though only one may be attacked.



FIG. 52.—Arthritis from Congenital Syphilis.

It is insidious in onset and, in spite of the severe degree of swelling, causes the patient no inconvenience during the day. When, however, he retires to bed there may be great discomfort on account of the characteristic night pains. This variety of syphilitic arthritis is frequently followed by interstitial keratitis.

Rarely syphilitic arthritis sets in acutely: the knees are the joints most frequently involved, and one joint may be attacked before the other. There may be fever, considerable pain and tenderness, so that confinement to bed is necessary. The arthritic exudate in this type is purulent in appearance from the abundance of polymorphonuclear leucocytes which it contains. It is easily seen how this variety of syphilitic arthritis may be mistaken for rheumatic arthritis, and it may be that a suspicion

of syphilis is only aroused when there is no response to salicylate therapy. Other evidences of syphilis are often wanting.

A degree of arthritis frequently accompanies syphilitic osteitis when this involves the articular end of the bone: here again pain may be little evident during the day, so that the child can jump about and play as usual, but the "night pains" are usually very disturbing.

On rare occasions a degree of multiple arthritis affecting the smaller joints of the hands and feet has a syphilitic basis; the importance of appreciating this fact is that anti-syphilitic treatment may give relief.

Acute Rheumatic Arthritis.—The age is an important element in the diagnosis of acute rheumatism. The disease never occurs before two, and very seldom before three, years old. Its onset is usually sudden, but it may be gradual. Occasionally only one joint is affected, and it is well to remember that this is, not very rarely, the hip-joint, when it is liable to be mistaken for appendicitis. Other indications of rheumatism are always to be looked for and asked about—especially heart affections, rheumatic nodules, erythema circinatum, chorea, and sore throat (see Chapter XXXV.).

In the early stage of slight cases there may be comparatively little limitation of movement. The child limps, however, when walking, and at rest keeps the joint flexed and still. There is much less redness, heat, and swelling than is usual in adults; if in any joint case in a young child these are marked features it is probable that the disease is not real rheumatism. The tenderness on handling may be considerable, although not nearly so bad as it usually is in adults. In true rheumatism pains in the fingers are common, but noticeable swelling of their joints is very rare. Often, according to the history given, the previous pains have been of a trivial nature, and have been regarded as merely "growing pains," or as the result of a sprain.

The pain is always rapidly relieved by the administration of salicylates.

Among the morbid conditions which we have seen mistaken for rheumatism the following may be mentioned: Still's disease, gonorrhœal arthritis, septic arthritis, acute infective epiphysitis, syphilitic epiphysitis, infantile scurvy, infantile spinal paralysis, tetany, flat-foot, and "pulled elbow."

Chronic Rheumatic Arthritis rarely occurs in children. It causes great stiffness from fibrous thickening of the joints, and there are always indications of rheumatic disease, past or present.

Rheumatoid Arthritis or Osteo-Arthritis.—This is rare in childhood, but it does sometimes occur—mainly in older children and adolescents. It resembles the adult disease in its clinical features, and, like it, is gradual in its onset and very chronic in its course. Several joints are affected and the distribution of the lesions is symmetrical. Bony changes occur in the later stages. The spleen and lymphatic glands are not enlarged; and there is no evidence of rheumatism or tuberculosis.

Still's Disease of the Joints (Chronic Arthritis with Enlargement of the Lymphatic Glands and Spleen).—This is a form of progressive polyarthritis which is much commoner in early childhood than ordinary rheumatoid arthritis. It is often regarded as merely an infantile variety of that disease, but Still¹ has given reasons for believing it to be a separate malady.

The disease often begins early—before three years old. We have seen more than one case which began at fifteen months. Generally the local symptoms are slight at first, though accompanied by debility and pallor. Sometimes, however, the onset is acute and febrile, and there may even be rigors. The joint affection consists in a fusiform enlargement of many of the articulations, and it is symmetrical in distribution (Fig. 53). The knees, ankles, and wrists are often involved, and affections of the cervical spine and of the sterno-clavicular and interphalangeal joints are especially characteristic. The capsule of the joint is thickened and feels pulpy, but there is little fluid effusion and no change in the bones. Suppuration never occurs. As the joints become swollen, the lymphatic glands of the affected limb usually enlarge considerably; and the spleen is also increased in size. The younger the patient, the more marked is the enlargement of the glands and spleen. The movements of the affected joints are sometimes little, if at all, limited in the early stages. Later the limbs become drawn up and the joints stiffen. The muscular wasting is very great. When the hand is placed on the joint during movement, slight crackling, connected with the tendons, is occasionally felt; but

¹ *Trans. Med. Chir. Soc. Lond.*, 1897, lxxx., 47.

there is generally little or no pain or tenderness. There is no tendency for this disease to be accompanied by endocarditis; but in many of the cases which have been examined post-mortem general pericardial adhesion has been found. The prognosis must be guarded, owing to the great debility and the consequent risk of intercurrent disease.

The treatment consists in life in a dry, warm climate, careful feeding, continued gentle passive and active movements of the joints. Hot salt-water baths and hot air-baths are sometimes also useful, and, internally, cod-liver oil, iodides, and arsenic.



FIG. 53.—Still's Disease of the joints in girl of 5 years.
(Dr John Playfair's case.)

The result of treatment, especially among the poorer classes, is often disappointing. The children are apt to become chronic invalids and to be carried off by intercurrent disease. Sometimes, however, care and good nursing result in more or less complete recovery. For example, a well-marked case, in a girl of four and a half, recovered completely after two years of treatment, partly at home and partly in Bath. Eleven years afterwards she was still quite well. A boy who had a very severe attack, beginning when he was six and lasting for many years, made, at last, a partial recovery. Twenty-two years after his first illness he was able to do regular work in a shop, although one hip-joint was quite ankylosed and his neck twisted and immovable.

Gonococcal Arthritis. — This condition, although not common, is occasionally met with in childhood. It may,

though rarely, occur in young babies with gonorrhœal conjunctivitis or vulvitis, as well as in older children. It sets in at any stage of the disease, but usually begins between the third and sixth week.

Generally only one joint is affected, and it is a knee in the majority of cases. The sheaths of some of the tendons are also frequently inflamed. The affected limb is kept flexed. The amount of swelling varies greatly in different cases; suppuration is rare. In older children the local condition may be very like that met with in acute rheumatism. The pain, however, is often strikingly severe, even apart from movement, and the way in which salicylates fail altogether to relieve it is an important point in the diagnosis.

Septic Arthritis.—Other forms of septic arthritis are met with occasionally in connection with many of the infectious diseases, such as scarlet fever, diphtheria, measles, enteric, influenza, erysipelas, and mumps. They also occur in connection with various general infections and local inflammations such as pneumonia, empyema, posterior basic and cerebro-spinal meningitis, otitis, and sore throat. Suppurative arthritis occasionally occurs in the new-born as a complication of an infected umbilicus.

There is usually a sudden onset during the course of the primary disease. Often more than one joint is affected, and abscesses elsewhere (for example, in the cellular tissue or parotid) are not uncommon. The temperature is usually high and remittent in type. The joint affection is not always acutely painful, and its course is sometimes subacute.

Spontaneous dislocation of the hip-joint, which occasionally occurs in cases of infectious disease, is to be accounted for by softening of the capsule of the joint from latent septic arthritis.

Joint Affections in Acute Poliomyelitis.—Occasionally acute joint swelling—especially of the ankle—is observed in cases of infantile spinal paralysis. This occurs usually at the onset of the disease and always recovers more or less rapidly. It is to be remembered because it may otherwise give rise to an error in diagnosis.

Hysterical Joint Affections.—When hysterical affections of the joints occur in childhood they are mostly found in older girls or boys; rarely, however, they are seen in quite young children. The onset is often sudden, and usually one joint only

is complained of. The patient is generally, though not always, a noticeably nervous and precocious child (p. 714).

The limb is held rigid by the spastic contraction of the muscles. Under chloroform, however, this disappears entirely—except in a few cases of very long duration. There is often distinct wasting of the muscles from disuse, even in not very long-standing cases. The affected joint is not swollen and looks altogether normal—the pain complained of being out of all proportion to the objective symptoms. There is frequently hyperæsthesia of the skin and other superficial parts. The pain is not increased by jarring movements of the joint. When the hip-joint is the part affected, the gait differs from that of ordinary hip-joint disease, and it is apt to vary in character from time to time. The lameness usually increases under observation.

It is advisable to be slow in committing oneself to a diagnosis of hysterical joint affection, and sometimes a positive opinion to that effect can only be given after prolonged study of the case. It may not, at first, be possible to distinguish between hysteria of a joint and early tuberculosis in a neurotic child. A careful observation of the temperature, and a tuberculin test may afford help in the diagnosis; and in doubtful cases an X-ray plate should always be taken.

Painless Effusion into the Knee-Joints.—Sir William Bennett has drawn attention¹ to a form of painless swelling of the knee-joints which is sometimes met with in young girls of twelve or thirteen or older, and less commonly in boys. It is in itself a trivial affection, but has some importance because it is apt to be mistaken for tuberculosis of the joint. In girls it seems to have some connection with the onset of menstruation, and sometimes the symptoms are so trivial that it escapes the notice of the patient herself as well as that of her mother, until some injury causes the joint to be specially examined. Both joints are usually affected, the right being often the larger of the two. In slight cases the effusion is only discovered when the lower part of the synovial membrane is made to bulge by the child's standing up. In others, a considerable amount of fluid is present; but the joint is never tense unless there has been a superadded injury. The patients are generally pale, nervous children, with various other complaints. When men-

¹ *Injuries and Diseases of the Knee-Joint*, London, 1909, 34.

struation becomes established the condition usually disappears. The treatment consists in careful attention to the general health, and an outdoor life with moderate exercise and gentle massage. The child must not be laid up, nor the limbs put in splints.

The Joint Affection of Hæmophilia consists in sudden effusion of blood into a synovial cavity—usually that of the ankle or knee-joint—along with stiffness, sometimes considerable pain, and a slight rise of temperature. When the joint affection is the first manifestation of the disease, as sometimes happens, its real nature is apt not to be recognised. The affected joint generally shows no discoloration on the outside, and its appearance is very like that of a tuberculous arthritis. It occurs, of course, only in boys. The treatment consists in rest and temporary fixation of the affected joint by means of a splint. Should the hæmorrhage recur, the joint is likely to be permanently damaged.

Articular hæmorrhages are sometimes also met with in cases of **Purpura**; and in **Erythema Nodosum** slight arthritis is occasionally found.

Occasionally, arthritis of moderate severity occurs in young infants, which does not conform to any of the above-mentioned types. The cases of the kind which we have seen have all recovered, and in none has there been any relapse or any permanent injury.

Schlatter's Disease (*Rugby Knee, Enlargement of the Tubercle of the Tibia*).—When asked to see a schoolboy with “a sore knee,” it is well to remember that the case may not be one of joint-lesion at all, but an instance of “Schlatter's Disease.”

This ailment consists in a swollen and tender condition of the tubercle of the tibia, with pain on movement, especially when the knee is extended as in going down stairs: there is no abnormality of the joint or of the bursæ in its neighbourhood. There may, or may not, be a history of a blow on the knee, or of special muscular strain having recently occurred. The condition is not a very uncommon one and may cause inconvenience and some anxiety; for, though the pain is not severe, it interferes greatly with active exercise and may be mistaken for rheumatism or for early tuberculosis.

An X-ray examination confirms the diagnosis and throws light on the pathology of the lesion. It shows that the downward prolongation of the upper epiphysis of the tibia, into

which the ligamentum patellæ is inserted, has been separated somewhat from the diaphysis behind it (Fig. 54). This dragging forward of the prolongation of the epiphysis may be present not only on the painful knee but also on the other side. It is evidently the result of muscular overstrain, and it constitutes the predisposing cause of Schlatter's disease. The exciting cause of the pain and tenderness may be a fracture, or

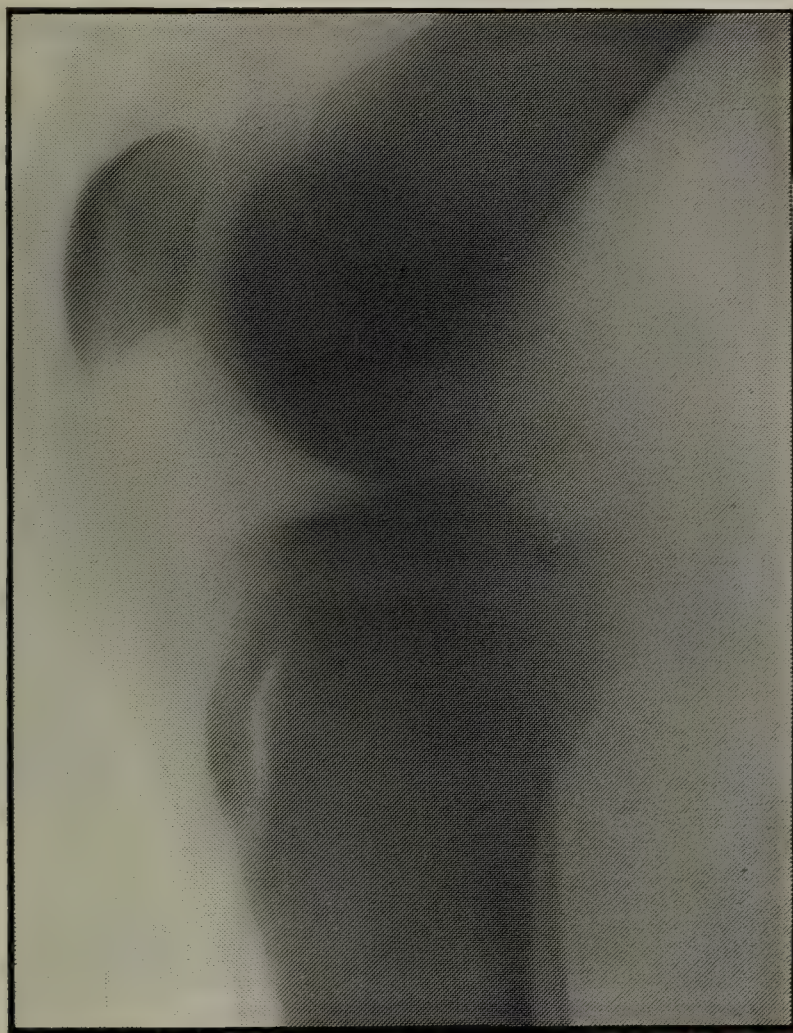


FIG. 54.—Skiagram of Schlatter's Disease in boy of 14 years.

other acute injury due to a blow or sudden pressure on the unduly projecting tongue of bone.

The treatment consists of rest in bed with a posterior splint, followed by a prolonged period of abstention from violent exercise. The pain often persists for many months in spite of treatment.

The Hands and Feet.

Examination of the hands and feet—especially of the former—should never be neglected. We may often gather useful information from them regarding the patient's general condition, besides noting any merely local abnormalities.

They may, for example, throw light on the state of the nervous system. In chorea the characteristic movements are seen in them as soon as anywhere. In tetany the attitude they assume is one of the most noticeable features of the disease. In young infants, constantly clenched fists are often a sign of great nervous tension, and may indicate that a convulsive attack is impending. The way in which the hand is held in certain attitudes of the arm varies in different states of the nervous system. A knowledge of these variations may be found of considerable use in the examination of school children as to their fitness for mental work. This has been worked out in an



FIG. 55.—Bitten Finger-nails.

interesting and original way by Dr Francis Warner in his various writings.¹

Inspection of the hands may reveal that the child habitually bites his nails (Fig. 55) or sucks his thumb. The recognition of such apparently trivial facts may not be altogether valueless in certain cases.

The state of the circulation is also seen in the hands and feet, as cyanosis and œdema show early in them.

Chilliness of the extremities is a common complaint in delicate and nervous children, especially in babies with rickets, and in older children who suffer from some degree of chronic intestinal dyspepsia. The symptom causes considerable discomfort and may give rise to griping pains in the abdomen. It is probably a cause as well as a result of dyspepsia.

¹ *Physical Expression*, London, 1885 ; *The Study of Children*, London, 1898 ; and others.

The treatment of cold feet and hands consists in regulation of the diet to suit the digestion, in seeing to the child's being sensibly clothed and having plenty of fresh air and exercise, and especially in the regular use of the cold douche with reasonable precautions (Chapter XXXVIII.). Occasionally small doses of thyroid may be useful.

Clubbing of the Fingers (Fig. 56).—The causation of this condition is still decidedly obscure. When it occurs, however, there is usually—probably always—some impediment to the



FIG. 56.—Clubbing of Finger-ends, from a case of Congenital Heart Disease.

circulation which is giving rise to engorgement of the systemic veins. The toes are usually affected as well as the fingers, and sometimes the nose and ears also. The presence of clubbed fingers is always important, and may sometimes help very much in the diagnosis of a lesion of the lungs or heart.

The *respiratory diseases* in which clubbing is oftenest found are phthisis, bronchiectasis, empyema, and chronic pneumonia. Clubbing of the fingers is more related to the severity of the lesion than to its duration. In young children it may be distinct within four weeks of the onset of the symptoms of the chest disease. If the latter recovers completely the fingers

will also soon become normal. The presence of clubbing may be useful in calling our attention to a latent phthisis or bronchiectasis which might otherwise have escaped detection.

In *congenital heart disease* severe clubbing is frequently present. Generally it accompanies cyanosis, but not always. It may not come on until the cyanosis has lasted for years, or it may never appear. Occasionally it is present when there is no cyanosis at all. When severe in degree it forms a bad element in the prognosis. Dr Lees¹ has pointed out that clubbing is more likely to accompany a congenital heart lesion when the foramen ovale does not remain patent, because under these circumstances there is more tendency to congestion of the systemic venous system. Older children who have severe and long-standing *valvular heart lesions* from endocarditis show only slight clubbing of the fingers. When the clubbing is severe, therefore, in young children with heart disease, it proves that the lesion is congenital in origin.

Clubbed fingers occur also sometimes in *hepatic cirrhosis*, *amyloid disease of the liver*, and *congenital hypertrophy of the colon*. A slight but distinct degree of clubbing is occasionally seen in delicate young children whose circulation is obviously defective but in whom no indication of any serious disease can be found.

The **conformation of the hands**, and to a less degree of the feet, may greatly help in the diagnosis of several diseases. As examples of this may be mentioned the characteristic hands of achondroplasia, which distinguish cases of that disease from those of dwarfing due to rickets and other causes (p. 872). The shape of the hands in mongolism, cretinism, and hypopituitarism is also an important diagnostic feature in these diseases.

The extent to which the finger-joints suffer in rheumatoid arthritis and in Still's disease helps to distinguish them from rheumatism (p. 84).

The Rickety Hand.—In many cases of rickets the hands are characteristically altered in outline (Fig. 57). The rickety hand is usually long and slender. The fingers show a tendency to spontaneous hyperextension and have a peculiar "beaded" appearance, the circumference of the joints being less than that of the middle of the phalanges. This beading, as the X-rays show, is due to elongation with narrowing of the joints and the

¹ *Trans. Path. Soc. Lond.*, 1880, xxxi., 58.

parts adjacent, and not to any actual thickening of the intervening diaphyses.¹

The presence of **tuberculous** or **syphilitic dactylitis**



FIG. 57.—Beaded Fingers, from a case of Rickets.

(Figs. 58 and 59) sometimes throws an important light on the causation of an obscure cerebral or abdominal case. These two conditions may be very like one another. The swellings of the fingers have a similar outline in both cases, and in both



FIG. 58.—Tuberculous Dactylitis.

they are usually multiple. In syphilitic cases, however, the metacarpal bones are less frequently affected and there is much less tendency to suppuration (Fig. 315, p. 925). The diagnosis

¹ Siegert, *Verhandl. d. Gesellschaft f. Kinderheilk.* (Cassel), 1903, xx., 240; Koplik, *Arch. of Pediatr.*, 1904, xxi., 771.

can generally be made from a skiagram, which shows the extent to which the interior and the surface of the bones are affected. In syphilitic cases the condition is one of gummatous periostitis, the interior of the bone being either unaffected or sclerosed; while in tuberculous dactylitis any periostitis present is more likely to be secondary to a caseous focus in the interior of the bone.



FIG. 59.—Congenital Syphilitic Dactylitis.

Deformities of the Thumbs and Great Toes in Myositis Ossificans.—These are described elsewhere (Fig. 262 and p. 856). They are of special interest because their recognition may lead to an early diagnosis of the commencing muscle change.

Arachno-dactyly or Spider Fingers.—These names have been given to an obscure congenital disease which was first described by Marfan,¹ and has more recently been dealt with by Thursfield,² Ormond and Williams,³ and others.

In a typical case observed by one of us (J. T.) in 1909 the chief symptoms were as follows: The patient, a big, fat, bottle-fed baby of six months who weighed $19\frac{3}{4}$ lb., was brought to the hospital on account of rigid flexion of his knees, elbows, and fingers, which had remained practically unchanged since birth.

His head was large (20 in. at 9 months), but not abnormal in shape, and, even when first seen, the fontanelle was closed. The ears presented the usual peculiarity in form which is characteristic of the condition—a prolongation of the crus

¹ *Bull. et Mém. de la Soc. Méd. des Hôp.*, 1896, 896.

² *St Bartholomew's Hosp. Rep.*, 1920, liii., 35.

³ *Guy's Hosp. Rep.*, Oct. 1924, lxxiv., 385.

helicis so as to form a well-marked ridge on the concha. The baby seemed to see quite well and nothing abnormal was noticed about his eyes, but unfortunately no ophthalmoscopic examination was made. In a majority of the published cases in which the eyes were properly examined there was found "congenital dislocation of the lenses with the concomitant symptoms of trembling irides, small contracted pupils, irregularly deep anterior chambers, and limited reaction to dilatation by atropin" (Ormond and Williams).

All the bones of the upper and lower limbs were abnormally slender and seemed rather longer than usual. The hands and feet appeared peculiarly long and narrow. Although the limbs were fat, their muscles were small and feeble; the knee-jerks were not obtained. The hip-joints, knees and elbows could not be extended fully; the fingers were also permanently flexed.

The baby was very shy and slow to make friends. He seemed to be rather backward, but showed no evidence of mental defect. While in the ward his weight and intelligence increased slightly; but there was no improvement in other ways.

Owing to their extreme muscular weakness, the children with this abnormality very often die early from intercurrent disease.

CHAPTER V

THE NOSE, THROAT, MOUTH, AND EARS

The Nose

A CHANGE in the *shape* of the nose is occasionally significant in diagnosis, especially the broadening and depression of the bridge which are found in many cases of congenital syphilis. Poorly developed and thickened alæ often accompany chronic nasal obstruction, and an exaggeration of the hollows above the alæ—"nasal dimples"—are usually, if well marked, an indication of the presence of adenoids, and are sometimes noticeable before the habit of mouth-breathing has been acquired.

Nasal obstruction, due to thickening of the mucous membrane by catarrh or other disease, is always suggestive of congenital syphilis when it occurs in a young infant, and of adenoids in older children; but acute nasal catarrh may be caused by streptococci, pneumococci, diphtheria bacilli, or other organisms. *A subacute or chronic nasal discharge should always arouse a suspicion of diphtheritic infection*, but a definite diagnosis can only be made from the finding of virulent diphtheria bacilli. A unilateral nasal discharge generally indicates the presence of a foreign body, such as a boot-button, a pea, or a bead in the nose. Polypi are rare and are only met with in older children.

When nasal obstruction sets in acutely, great distress may result, especially in young infants, because it prevents the child breathing comfortably through the nose. Besides causing local discomfort and headache, it leads to serious interference with the free entrance of air into the chest, so that it may constitute a dangerous complication in such conditions as pneumonia and acute bronchitis. In these, attention to the nasal obstruction is always an important part of the treatment.

Catarrhal obstruction of the nasal passages may generally be greatly relieved by the frequent use of a simple alkaline cleansing

lotion (sod. bicarb. 10 gr., ac. boric. 10 gr., sod. chlorid. 3 gr., and water 1 oz.). This should be warmed to the temperature of the body and may be sniffed into the nostrils from a teaspoon, or gently injected by a medicine dropper.

Rubbing the Nose.—One of the early symptoms in guinea-pigs and other animals that are suffering from anaphylaxis is a violent rubbing of the nose, with sneezing and sniffling. The same symptoms are often seen in little children with asthma, and also in those who have an idiosyncrasy for eggs or other kinds of food.

Epistaxis.—Nose-bleeding is a common symptom in childhood. It may be due to a passing cerebral congestion, to acute nasal catarrh, to the child having picked off a small scab near the front of the septum, to a foreign body, or to a blow on the nose. The presence of adenoids predisposes to its occurrence. In the majority of cases bleeding from the nose is a matter of no importance. Sometimes, however, it occurs as a symptom of some form of serious disease. It is not uncommon, for example, in congenital heart-disease, at the beginning of an attack of acute rheumatism, in enteric, in some epidemics of influenza, in chronic nephritis, and in such blood diseases as leukæmia, purpura, and hæmophilia. It may, in rare cases, be a precursor of hay fever. When epistaxis occurs in a child who is lying down, the first sign is usually the vomiting of the blood which has been swallowed, and this may be mistaken for gastric hæmorrhage.

To stop the bleeding, the child should be made to stand up with the arms raised and the chest expanded, while the feet are kept warm and cold cloths applied to the nose. In obstinate cases a small plug of cotton wool soaked in peroxide of hydrogen should be placed inside the nostril and pressed inwards. The bleeding point is almost always situated near the front of the septum. Plugging of the nares is rarely, if ever, required.

The Mouth and Throat.

The examination of the mouth and throat includes the inspection—and sometimes the palpation—of the lips, tongue, teeth, gums, cheeks, palate, tonsils and fauces, pharynx, nasopharynx and larynx.

It is extremely important that the mouth and throat should

always be thoroughly examined in all cases of feverish illness in childhood, even if no local symptoms are complained of. Many cases of obscure pyrexia are cleared up at once by such an examination.

Methods of Examination.

Inspection.—It is generally easy to induce a baby who is not frightened, and is not teething, to open his mouth if you gently touch his lips with the finger; and, once the mouth is open, the finger can readily be passed along the gums till it touches the pharynx, thus compelling the child to give a good view of the fauces.

The main points to be attended to in examining the throat are, first, to have the child facing a good light in such a way that, when the mouth is opened, the fauces will be illuminated at once without any change of position; and secondly, to have his arms held, or secured to his side by a towel pinned round him, so that he cannot suddenly seize the tongue-depressor or the hand that holds it. If the examination of the throat is carried out rapidly and gently the first time and no struggling allowed, it will be much easier on the next occasion; but if the child is allowed to struggle it will be more difficult each time. An electric torch is a very useful aid in the examination of the mouth and throat.

Digital palpation of the fauces, pharynx, and naso-pharynx is especially important in young children, because it is often so difficult in them to get a good view of these parts and, with practice, much information can be got from it. Without palpation it may be impossible to make sure of the presence or absence of a retro-pharyngeal abscess or of adenoids.

When the throat is examined with the forefinger in older children, the cheek should be pressed between the side teeth by the fingers of the other hand, so as to prevent the examining finger being bitten; a gag is usually unnecessary.

The Breath.

A healthy child's breath is, of course, free from odour of any kind, but this is often changed in illness. Occasionally, for example, the breath has a distinct smell of acetone, the significance of which is considered elsewhere (p. 551); and often, for one reason or another, it is decidedly offensive.

Foul Breath.

In the course of severe lung diseases, such as pneumonia or pulmonary tuberculosis in delicate children, a putrid smell of the breath may suddenly appear. This is a grave symptom, as it generally means the onset of gangrene of the lung. Under ordinary circumstances an offensive breath has a much less serious significance; still, it may occasion so much distress and discomfort that the recognition and treatment of its cause are matters of considerable importance.

In children with carious teeth or pyorrhœa, ulcerative stomatitis, unhealthy tonsils with decomposing matters in the crypts, atrophic rhinitis, or bronchiectasis with ill-smelling sputum, a foul breath is a common and easily explained symptom, and the indications for its treatment are more or less obvious. Sometimes, also, an offensive odour occurs in children with a coated tongue and other indications of digestive derangement.

The most perplexing cases, however, are those in which, although the breath is distressingly offensive to the patient and his friends, no apparent cause can be discovered. The children are often, in other respects, in good health. It has been suggested that in these cases some foul smelling organic matters derived from the food, which should have been dealt with by the liver and bowel, have escaped these purifiers of the system, and are being excreted by the glands of the upper digestive and respiratory mucous membranes instead. Some of the cases are very intractable, but many of them yield rapidly to the regular use of simple measures.

Treatment.—The following treatment may be tried :—

(1) The amount of food taken must be confined to normal proportions; these children are often very large eaters.

(2) The proportion of sapid substances in the food should be specially limited.

(3) Plenty of open-air exercise must be insisted on.

(4) The bowels should be kept freely open by a daily morning dose of Carlsbad salts or mineral water.

(5) Rhubarb and soda may be given twice daily, and a dose of calomel once every week or ten days.

(6) Charcoal lozenges or biscuits and a weak permanganate of potash mouth-wash are also sometimes useful.

The Lips.

Pallor of the lips forms, in children as in adults, a fairly trustworthy sign of anæmia ; and a slight purplish tinge is often of importance in heart and lung cases as an indication of commencing cardiac failure.

Eczema and catarrhal herpes of the lips are not uncommon in young children, and several forms of stomatitis, such as the aphthous, syphilitic, and diphtheritic varieties often affect them severely. During the onset of measles the inside of the lips may show Koplik's spots, though their commonest site is the inside of the cheeks near the first molar tooth.

Cicatricial fissuring of the lips, as has been elsewhere noted, is an important sign of congenital syphilis, and a slighter degree of fissuring, confined to the mucous membrane, is seen in many cases of mongolism.

Dribbling of saliva from the mouth is met with under various conditions. It is normally present in most babies during teething, especially when there is any irritation of the gums from stomatitis. Sometimes it sets in during acutely painful conditions of the throat which make the child unwilling to swallow his saliva ; and it is a characteristic early symptom in spasmodic stricture of the œsophagus in which the saliva is greatly increased in amount. It is also a very common and significant symptom of mental defect (p. 811).

The Tongue.

In new-born children the mucous membrane of the mouth is of a dark red colour, and for the first few months it is noticeably dry owing to scantiness of the saliva. For the same reason the tongue is apt to be more or less coated in young infants.

It is not uncommon in children to find red areas on the dorsum covered with thinned epithelium and bounded by white or greyish elevated margins which have an irregularly rounded contour. When this condition is extensive its irregular outlines resemble those of a map, and hence it is spoken of as the "mapped" or "geographical tongue" (Fig. 60). Usually there is no local pain or discomfort in these cases, and no apparent digestive disturbance. It is often found in children who have

the so-called "exudative diathesis"¹; it has no connection whatever with congenital syphilis, and is certainly not a tuberculous lesion. In the rare cases in which there is pain it is best treated with krameria lozenges.

Tongue-tie is often complained of by mothers, but is seldom found present to such an extent as to warrant operative interference. When, however, the frænum is so short that the



FIG. 60.—"Geographical Tongue."

tip of the tongue is turned downwards when an attempt is made to protrude it, it should be divided, as it may possibly interfere with sucking in infancy and in after life with articulation.

A small erosion or *ulcer* is occasionally found *below the tip of*

¹ "Exudative diathesis" is a term first employed by Czerny for a supposed congenital, hereditary, and familial tendency to inflammatory conditions of the skin (eczema) and catarrh of the mucous membranes (naso-pharynx, bronchi, and alimentary tract). (See A. Czerny, *Jahrb. f. Kinderh.*, 1905, lxi., 199; A. Czerny and A. Keller, *Handbuch d. Kindesernährung*, Leipzig, 1917, ii., 352.)

the tongue (Fig. 61) and often on the margin of the frænum. This almost always indicates the presence of whooping-cough, during the spasms of which the tongue is frequently shot out over the sharp lower incisors. It may, however, occur in any form of violent cough if the teeth are sharp; and even, rarely, without cough, in cases where the movements of the tongue in sucking are very energetic.

The *Strawberry tongue*, which is characteristic of scarlet fever on the third day and onwards, is sometimes a great help in the diagnosis of that disease. It is well, however, to

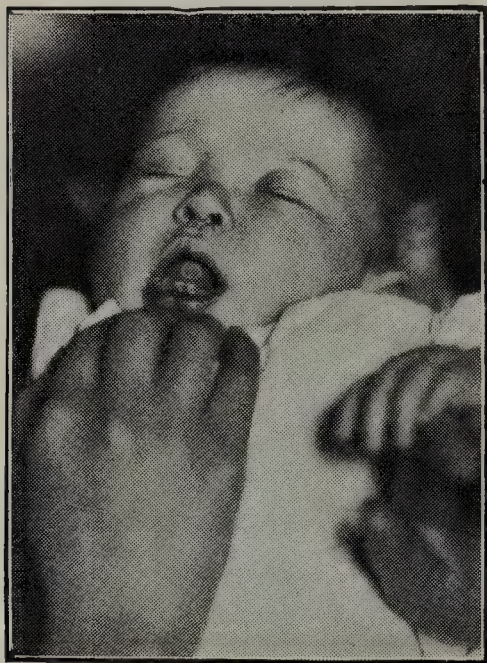


FIG. 61.—Infant with Whooping-cough and Congenital Syphilis. Ulcer under tongue. œdema of eyelids, depressed bridge of nose, and nasal discharge.

remember, not only that this appearance is often absent in scarlet fever, but also that a typical strawberry tongue is occasionally seen in other conditions.

Fissuring of the Tongue, as mentioned elsewhere (p. 832), is extremely common in mongolism. This is mainly due to the vigorous and persistent sucking of the tongue which these children practise. It is also met with not very rarely in cretins and in other children, whether mentally defective or not, if they are addicted to sufficiently continuous sucking of the tongue.

Tongue-swallowing (*Aspiration of the Tongue*).—In some delicate babies there is a tendency to choke from the tongue falling right back into the pharynx and so obstructing the opening of the glottis. For this to occur there must be a flabby tongue with a long frænum and generally loose attachments. It is probably never caused by too free division of a tight frænum.

We have seen aspiration of the tongue occur in the paroxysm of whooping-cough, and it also occurs in laryngismus. It is favoured by the presence of nasal obstruction from catarrh. One of the worst and most persistent cases of the kind I have seen occurred in a wasted baby with subacute posterior basic meningitis. In it the parts were so relaxed as to appear paralysed. Aspiration of the tongue is an occasional cause of sudden death in young infants.

The Teeth and Gums.

The examination of the teeth has been already discussed (Chapter III, p. 49), and stomatitis, as it affects the gums, will be considered later.

The Cheeks.

Sucking-pads.—An interesting peculiarity in young children is the prominence of the cheeks caused by the sucking-pads.¹ These are little separate encapsulated masses of fat which lie outside the buccinator and masseter muscles and prevent the



FIG. 62.—Sucking-pads in a child of 7 months.

falling in of the cheeks during the normal process of sucking. As emaciation proceeds, these pads generally waste more slowly than the surrounding adipose tissue. This results in the appearance on each cheek of a rounded projection, like half a small marble, which gives a peculiar look to the child's face, especially when he cries (Fig. 62). These projections are frequently seen in children under two years who are wasting rapidly from any cause, such as diarrhœa. They are much less common in older children. We have, however, seen them very well marked in a boy of eleven years with diabetes.

¹ Ranke, *Virchow's Archiv.*, 1884, xcvi., 527; Lehndorff, *Jahrb. f. Kinderheilk.*, 1907, lxvi., 286.

The Palate.

Epithelial Pearls.—In many infants during the first few weeks little yellowish white rounded nodules, from the size of a pin-head to that of a millet seed, may be seen about the mesial line of the hard palate embedded in the mucous membrane. They are usually called *epithelial pearls*, being composed of collections of epithelial cells, and they have generally no clinical significance. In unhealthy infants, however, they occasionally ulcerate, and when unusually large may give rise to unnecessary anxiety from being mistaken for manifestations of congenital syphilis.

Cleft palate may be responsible for difficulty in sucking and for regurgitation of food through the nose. The mildest degree of this malformation is a bifid uvula, which is not infrequently observed.

Chronic *ulcers* are not uncommon towards the back of the hard palate in wasted babies of a few weeks. They are caused either by the nurse's finger, which has been too roughly used in cleansing the mouth, or by the continuous pressure of some hard body which the baby has been allowed to suck. They often recover in a week or two under weak boric applications, but sometimes they are very obstinate. These ulcers are often referred to as "*Bednar's aphthæ*."

Abnormal vaulting of the palate may be found even in early infancy, but the deformity is rarely very great until after the second dentition. Such peculiarities, however, are of little importance from a practical point of view.

Perforation and destruction of the soft palate are generally due to congenital syphilis or to lupus. Perforations of the pillars are occasionally met with in Vincent's angina.

Paralysis of the soft palate is very characteristic of post-diphtheritic paralysis, and hence in this disease is an important diagnostic feature.

The palate should be carefully examined in cases of suspected measles, as the rash may appear there from twenty-four to forty-eight hours before it is recognisable on the skin. The rashes of scarlet fever and chicken-pox are also often seen in this position.

Stomatitis.

Various forms of inflammation of the mucous membrane of the mouth are common in childhood, and they are of considerable importance for several reasons. They are apt to be overlooked, and often give rise to a surprising degree of fever and general disturbance. They may also occasion great distress and interfere very much with chewing and swallowing, and consequently with the nutrition of the child. The main varieties of stomatitis are as follows:—

1. **Catarrhal Stomatitis.**—Catarrhal stomatitis may be local or general. When local, it is usually due to the irritation of decaying teeth; when general, it may be caused by a dirty teat or “comforter.” It is commoner during teething than at any other time.

The symptoms consist in swelling, pain, heat, and redness of the mucous membrane of the mouth, with a rise of temperature (sometimes even to 104°), and other signs of febrile disturbance. The tongue is covered with a thick yellowish white fur. There is generally also an increased secretion of saliva which runs out of the mouth, and there may be some enlargement and tenderness of the lymphatic glands below the jaw. The child often takes the breast or bottle eagerly at first, but stops very soon and cries with pain and annoyance.

The *treatment* consists in giving cold food from a spoon if sucking is painful, and in swabbing the mouth with peroxide of hydrogen or a saturated solution of boric acid after each feed. In obstinate cases the mucous membrane should be painted once daily with nitrate of silver solution ($\frac{1}{2}$ to 1 per cent.), and 2 or 3 gr. of chlorate of potash given internally every three or four hours. In older children, lozenges containing formaldehyde are also useful. Recovery usually takes place in about a week.

2. **Aphthous Stomatitis.**—The etiology of aphthous stomatitis is still unknown, although various organisms have been described as its cause. It is regarded by many authorities as not contagious, but we frequently see it in several members of a family at the same time.

The aphthæ appear as small rounded ulcers, which begin as vesicles and usually have a little yellowish exudation about their edges. They vary greatly in number in different cases,

and the amount of general stomatitis which accompanies them also varies. If the general stomatitis is severe there is usually a considerable degree of fever (102° to 103°). In some cases the ulcers are accompanied by little redness of the intervening mucous membrane, and in these cases there may be no general disturbance; but a good deal of local tenderness and consequent interference with the feeding are frequently present.

The local application of hydrogen peroxide, boroglyceride or permanganate of potash (1 to 1000), along with the internal administration, three or four times daily, of chlorate of potash (2 gr.) and tincture of the perchloride of iron (2 to 5 minims according to age) is usually rapidly followed by recovery.

3. Parasitic Stomatitis.—Parasitic stomatitis or thrush is due to the growth on the mucous membrane of an organism related to the yeast fungus (*Saccharomyces albicans*). This organism occurs in the mouths of many healthy children, but stomatitis only results when the mucous membrane has been damaged. This is almost always in consequence of the habit of cleaning the mouth with a piece of gauze or linen on the finger of the nurse. In the normal infant before the teeth appear the tongue is an efficient cleanser of the mouth. Thrush is seldom seen in babies whose mouths have not been artificially cleaned.

At first the disease causes small white raised spots on the tongue and on the inside of the cheeks. These look like fragments of milk curd, but can only be removed by using some force, and leave an abrasion behind. In severe cases the spots spread till they form a continuous false membrane which may extend to the throat, or even in rare cases down the œsophagus. When a portion of the white patch is treated with liquor potassæ and examined under the microscope, it is found to consist of the filaments and spores of the fungus along with epithelial cells, milk globules, and bacteria. There is usually catarrh of the mucous membrane between the patches.

The *treatment* in the milder cases consists in the cessation of all manual interference with the mouth and the instillation of glycerine and borax. In the severe forms the spots should be removed very gently by means of a cotton swab moistened with bicarbonate of soda solution (1 drm. to 5 oz.), and applying

permanganate of potash solution or glycerine and borax to the raw surface. Everything that comes near the child's mouth, or has to do with his milk, must be sterilised so as to avoid reinfection, and attention must be given to the causes of the general debility.

4. Ulcerative Stomatitis. — This variety does not occur before the appearance of the teeth, and in the vast majority of cases is closely related to caries. Although the condition may be predisposed to by defective nutrition and the exanthemata, such as measles, it not infrequently occurs in well-nourished vigorous children. Ulcerative stomatitis is apt to occur in mercurial, lead, and phosphorus poisoning, and it is also sometimes a symptom of scurvy. It is most frequently met with between five and ten years of age.

The ulceration begins at the seat of a carious tooth at the alveolar margin and spreads thence to neighbouring parts. Sometimes the teeth are loosened and the jaw may even become necrosed. The lymphatic glands are enlarged, and there is a constant dribbling of saliva, which is frequently blood-stained. The tongue presents a dirty brownish-yellow fur and the breath is fetid. The bacteriology of the condition is not thoroughly understood, but fusiform bacilli along with spirilla have been described and also an amoeba (*Entamoeba gingivalis*).

In cases due to scurvy or poisoning the treatment of these conditions is, of course, the main thing. The special treatment consists in giving chlorate of potash (2 gr.) with tincture of perchloride of iron (2 minims or more) internally every two or three hours; and if the child is cachectic, opium is sometimes very useful. The mouth should be painted frequently with permanganate solution (1 to 1000). The patient's nutrition and his hygienic surroundings must be improved, if possible, and precautions taken against his infecting other children. Carious teeth may have to be removed.

5. Gangrenous Stomatitis. — Cancrum oris or noma is fortunately not a very common disease. It only occurs in children whose vitality has been seriously lowered by profound malnutrition or by one of the infectious diseases, especially measles; and it is probably not infectious to healthy children.

Many micro-organisms have been found in these cases, but their exact relationship to the causation of the disease has not

yet been determined. Recently fusiform bacilli with spirilla have been described as characteristic.

The inflammation begins as a small red patch on the gum, or on the cheek near the angle of the mouth. This rapidly



FIG. 63.—Cancrum oris following Measles. (Dr Harvey Littlejohn's case.)

spreads and soon assumes the characteristics of moist gangrene, destroying all the tissues affected (Fig. 63). In the majority of cases the child dies in a state of collapse, or with symptoms of septicæmia or pneumonia. Occasionally recovery takes place with, or rarely without, active treatment, and great

deformity is always left. In girls, noma is sometimes met with on the vulva.

The treatment consists in a thorough application of Pacquelin's cautery, pure carbolic acid, or some other escharotic; and opium should be given in full doses.

6, and 7. **Diphtheritic** and **Syphilitic Stomatitis** need not be specially described here.

8. **Other Forms of Infective Stomatitis.**—One severe case of pseudo-membranous stomatitis in R.H.S.C., Edinburgh, which was due to a streptococcus, after resisting all other treatment, recovered rapidly when treated by injections of anti-streptococcal serum.¹ Another case which looked very like one of diphtheria was found to contain a form of leptothrix; in it the application of peroxide of hydrogen was very efficacious.

Catarrhal Sore Throat.

Ordinary sore throat may be present at any age and is common in infants as well as in older children. In infancy its presence is sometimes shown only by pyrexia, but often the child also refuses his food and shows signs of pain on swallowing. In older children, slight degrees of catarrhal sore throat often give rise to a persistent cough as the most prominent symptom.

The recognition of acute sore throat is generally easy if the fauces are inspected; but, especially if fever is present, its presence may indicate the beginning of one of the infectious diseases, such as scarlet fever, diphtheria, enteric, rubella, measles, or influenza.

Acute tonsilitis is not uncommonly a rheumatic symptom, and the heart should always be watched during the attack for any signs of cardiac dilatation or endocarditis. When several children in a household suffer from sore throat the condition of the milk and water supply should be investigated.

The *treatment* of the condition in children does not differ from that in adults. In acute cases it is generally well to begin with a mercurial purge, and this has often a decided effect in relieving the pain in the throat. Chlorate of potash may also be given, and in possibly rheumatic cases salicylate of soda. The frequent recurrence of sore throat generally

¹ J. S. Fowler, *Arch. of Ped.*, 1901, xviii., 356.

indicates the necessity of an operation for enlarged tonsils or adenoids. Whether these conditions are present or not, the recurrence may sometimes be prevented by the administration of an autogenous vaccine.

Vincent's Angina.

Vincent's angina is a mildly infectious form of ulceration of the throat, mouth, and gums, which is characterised by the formation of a false membrane, and is apt to be mistaken for diphtheria. There may be deep ulceration of the tonsil, and the false membrane sometimes spreads on to the palate and uvula; perforation of the pillars of the fauces is much commoner than in diphtheria. The disease is often confined to one side. The fever and other constitutional symptoms only last for a few days and are not severe; the glandular enlargement does not tend to suppuration.

Scrapings from the surface of the lesions show a mixture of fusiform bacilli and large spirilla, similar to those found in ulcerative stomatitis and in noma. The diagnosis depends mainly on the presence of these organisms; but the depth of the tonsillar ulceration, the patches on the gums and cheeks, and the mildness of the symptoms are so far characteristic.

The prognosis is good, as extension to the larynx and trachea is very rare. The lesions, however, sometimes react slowly to treatment and may take several weeks to heal.

The treatment consists in the assiduous application of hydrogen peroxide or some other antiseptic, and in the repeated irrigation of the mouth and throat with sterile water or saline solution for ten minutes at a time. An arsenical lotion (Appendix E, Form. 39) as a local application is recommended by Dr Donald Paterson.

Chronic Enlargement of the Tonsils.

Enlarged tonsils are usually, though not always, associated with adenoids. There is much difference of opinion regarding what should be considered an abnormal tonsil. In our opinion any tonsillar tissue visible beyond the pillars of the fauces is abnormal and calls for removal. It must not be forgotten, however, that some of the most diseased tonsils are deeply placed and are scarcely visible.

Enlarged tonsils are apt to be inflamed and lead to otitis media. They are a frequent source of cervical adenitis; they also interfere with breathing and lead to recurrent attacks of catarrh of the larynx and bronchi. They are also supposed to be the chief portal of entry of the rheumatic virus, and certainly it would seem that arthritis is less common in children who have had them removed. In not a few cases they are the cause of the child's having a fetid breath and a constant furred tongue because of the septic material which they harbour.

Adenoid Growths.

Even in infants of a few months adenoids may be present, and they are very common at all ages. The change which their presence produces in the face has been already referred to (p. 11), and their effect on the form of the chest is also described elsewhere (p. 601).

In *young infants* our attention is drawn to the condition chiefly by signs of local obstruction. There is usually mouth-breathing, often impairment of hearing, sometimes otorrhœa, obstructed respiration with snoring, or with a sort of croaking which may be mistaken for congenital laryngeal stridor (p. 655), and, occasionally, some interference with taking the breast. In very young babies there may be no recognisable adenoid facies at first, because it is some time before the child learns the relief of breathing through the mouth. There is often a history of recurrent bronchial catarrh.

In *older children* one of the most important symptoms is deafness, which may, or may not, be accompanied by recurrent attacks of middle-ear catarrh. Headache is common and the voice is noticeably nasal in tone. The child generally sleeps with his mouth open, and snores. In some cases there are frequent attacks of bronchitis, which usually begin with a sore throat; in a few there are asthmatic symptoms; in others a peculiar choking paroxysmal cough which is apt to be mistaken for whooping-cough. When there is much naso-pharyngeal obstruction night terrors often occur, and enuresis is not uncommon. General debility and languor are present in many cases.

The term "*Aprosexia*" has been applied to a peculiar type of mental dullness which is common in children with much nasal obstruction from adenoids. The exact causation of the

condition has not been determined, but it looks as if the blocking of the circulation through the nose and naso-pharynx interfered in some way with that of the frontal lobes. The children are usually quite bright and lively at times; but often, especially when the mucous membranes are swollen from catarrh, they become dreamy, absent-minded, and timid to such a degree that their teachers may suspect them of being mentally defective. The connection between these symptoms and the adenoids is proved by the rapid and striking improvement which follows the removal of the latter.

Treatment.—If the presence of adenoids is recognised early, at a time when the first signs of the disease are beginning to appear, much improvement may probably be obtained in many instances by the persistent use of simple medical measures—and complete recovery in some. At present, however, the great majority of the cases which are brought to the physician on account of adenoid symptoms already show the disease in such an advanced form that operation is certainly advisable.

Medical Treatment.—It has been the experience of most physicians that, occasionally, cases in which the operation has been postponed, and in which the health has in the meantime improved greatly under general treatment, have ended in complete recovery. Much has also been written about the efficacy of certain simple breathing and other exercises (suggested by Mrs E. Handcock) in restoring the naso-pharynx to a healthy condition, or at least lessening the obstruction.¹

The treatment recommended consists in regular drill and breathing exercises in a well-aired room. The children are taught to make a series of forcible inspirations, alternating with vigorous blowing of the nose, carried out in such a way that the nostrils are not compressed during the act. After this has been repeated about ten times, sneezing is induced by the application of a mildly irritating powder (iris root and soap) to the anterior part of the septum just inside the nostril. The sneezing is continued until the passages are clear and a “dry sneeze” results; and, thereafter, the inspirations and blowing of the nose are repeated, first with both nostrils and afterwards with one at a time. It is also well to cleanse

¹ See J. Ormiston, *Lancet*, 1918, ii., 240; Gertrude H. Hickling, *Brit. Med. Journ.*, 1920, i., 147; and Alex. Fraser, *Brit. Med. Journ.*, 1920, i., 310.

the passages further by sniffing up an alkaline and antiseptic lotion (*e.g.* ac. boric. and sod. bicarb. āā 10 gr., sod. chlorid. 3 gr., water 1 oz.); and the child should be encouraged to keep the mouth closed habitually and to breathe through the nose.

Several school medical officers, who have organised classes for the practice of methods such as those above mentioned, have reported encouraging results, and it seems likely that more improvement in the local symptoms may be effected in this way than was formerly thought possible. The general health and digestion are said also to benefit remarkably.

It is probable, therefore, that medical treatment of this kind is well worth trying in early cases and in those where the general health is good, provided there are no serious facial or thoracic deformities, and no indications of chronic or recurrent affections of the ears or of the respiratory or digestive organs. They may also be made use of when the symptoms are moderate in degree and have only set in recently during an attack of infectious disease, or as the result of some general or local condition which is likely to yield to treatment.

Surgical Treatment.—There are several important indications for surgical treatment in cases of adenoids; and what there is to be said on the subject applies equally to the subject of removal of the tonsils. In the *first* place, an operation is necessary whenever the *local obstruction* due to the growths is obviously preventing the free entrance of air through the nasal passages, because we know that a continuance of this will certainly result, if it has not already done so, in deformity of the features, an unpleasant change in the voice, and an alteration in form of the thorax which will prevent its full and free expansion in respiration, and therefore be detrimental to the general health. If the respiratory obstruction develops in infancy, the early removal of its cause is even more urgently called for, because it may also interfere with the child's nutrition by preventing his taking the breast or bottle with comfort.

There are, *secondly*, in a few cases, reflex nervous symptoms which seem to be due to the presence of adenoids and which sometimes, though not always, cease on their removal. Among these are laryngeal spasm, paroxysmal coughing, asthmatic attacks, habit spasm, and nocturnal enuresis.

The *third* indication for operation—and a very important

one—is the recurrence of attacks of certain acute diseases of an infective nature. The naso-pharyngeal adenoid tissue is peculiarly liable to bacterial invasion of all sorts, and forms a ready nidus from which infection spreads to neighbouring parts. This may occur even when the adenoid hypertrophy is moderate in degree.

Examples of the common types of infection starting from adenoids are found in such recurrent conditions as nasal catarrh, sore throats, middle-ear inflammation, and catarrh of the trachea and bronchi. The routine attention to the throats of school children has brought about a great diminution in the number of cases of deafness due to otitis media. We often also find indigestion, pallor, and general debility which owe their origin to the constant swallowing of septic naso-pharyngeal secretions. In rheumatic children the recurrence of such catarrhal affections is especially dangerous. When they have adenoids, therefore, an operation should always be done.

In some instances the parents are strongly opposed to any surgical procedure; but, if the child is otherwise healthy, there is little ground for this. The very large experience of such operations, which has accumulated in the course of the last thirty years, has shown conclusively that the results are entirely satisfactory in the great majority of cases, that the risk is very small, and that the serious consequences that have been predicted by some writers practically never occur. It has, for example, been suggested that the child's voice in singing may be permanently injured. When this occurs, however, it is probably always due to the operation having been done badly, so that the pillars of the fauces or the soft palate have been torn.

It is important that, after the operation, attention should be given to the child's breathing, and that he should have regular exercises to favour the development of his chest as much as possible.

Retro-Pharyngeal Abscess.

In examining children with dyspnœic symptoms or sore throat—especially those under two—it is important to remember the possibility of retro-pharyngeal abscess. It is not a common condition, but its recognition and proper treatment are of great importance.

Chronic abscesses in this situation are often the result of *tuberculous disease* of the cervical vertebræ, and the pus is situated, at first, between the bone and the prevertebral fascia. In *acute* cases the abscess develops in front of the prevertebral fascia and pushes the posterior pharyngeal wall before it, so that it bulges into the back of the throat, interfering with deglutition and respiration. It is generally due to infection of the lymphatic glands by *pyogenic organisms* from the throat or elsewhere. It is the acute or subacute cases to which the following remarks apply.

The onset of the *symptoms* is usually insidious. The child is restless, and sometimes refuses his food, and seems pained when drinking. There may be some stiffness of the neck. The breathing early assumes a snoring character, especially when the child is asleep; but generally it is ten or fourteen days before the abscess is sufficiently large to cause interference with breathing. Gradually the respiration becomes more and more difficult, and stridor accompanies both inspiration and expiration; and the breathing is worse when the child is laid down. When the patient attempts to drink he is apt to choke, and the fluid is coughed out of his mouth and nose. Generally neither hoarseness nor cough is present, but this is not always so, as the condition may be accompanied by laryngeal catarrh. Occasionally there is a definite external swelling in the neck. The only certain means of diagnosis, however, is *digital exploration* of the pharynx, which at once reveals the presence of the abscess, even when it cannot be recognised by inspection.

The *treatment* in acute cases consists in immediate incision. This is best done through the mouth by means of a tenotomy knife, the child being turned on his face immediately after the incision, so that he may cough out the pus. Chronic abscesses should usually be opened through the neck.

The Ears.

In infancy and early childhood *acute otitis media* is fairly common, but it is often overlooked because in so many cases at these ages it causes no complaint of local pain. It is responsible for many examples of apparent nutritional disease in infancy, whether there is or is not fever. Consequently it is important that the ears should be examined in all babies in whom the

cause of the disease is uncertain, and particularly in examples of unexplained pyrexia, more especially when there are symptoms suggestive of meningitis. *Chronic ear disease*, however, is not nearly so frequent in infants as in older children.

With the electric otoscope the ears can now be satisfactorily examined in the youngest infant. Mistakes will be made, however, unless it is remembered that during infancy the tympanic membrane is more vascular and less translucent than during later life, so that the ossicles and the cone of light are seldom visible. The least contact with the membrane, too, induces hyperæmia, and crying may cause quite definite bulging. Localised bulging in the upper and posterior quadrant (Shrapnell's membrane) or a dull greyish appearance of the membrane are the most definite evidences of pus in the middle ear.

Deafness during the first year or two of life is usually the result either of a congenital defect, or disease of the ears or brain (congenital deaf-mutism)¹; of labyrinthitis; of meningococcal meningitis damaging the internal ear; or of otitis media—simple, congenital syphilitic, or tuberculous.²

During the early years of school life ear discharges and impaired hearing become extremely common. This is due to the frequency with which chronic inflammations of the throat and naso-pharynx follow attacks of scarlet fever, measles, diphtheria, whooping-cough, and influenza, and spread from one child to another in school.

Such infections often lead to serious diseases which in some cases endanger life, and in others leave permanent deafness. There is indeed good reason to believe that nearly all the chronic middle-ear disease of later life—suppurative and non-suppurative—(with the exception of otosclerosis) originates during childhood. It is therefore essential that all cases of otorrhœa should be dealt with early and very thoroughly.

The commonest morbid conditions found in chronic deafness in young school children—apart from deaf-mutism—are collections of wax, enlarged tonsils and adenoids, chronic middle-ear inflammation and mastoid disease.³

¹ J. S. Fraser, *Journ. Laryngol. and Otol.*, Jan. 1922, 13.

² See Chap. XXXVII., p. 983.

³ J. Kerr Love, *Diseases of the Ear in School Children* (J. Wright & Sons, Ltd.), 1919; A. Logan Turner, *Diseases of the Nose, Throat, and Ear* (J. Wright & Sons, Ltd.), 1924.

Treatment.—In *acute suppurative otitis media* the child should be confined to bed and a dose of calomel given, followed by a saline purge. A few minims of carbolic acid and glycerine (℥ v—℥ i) should be dropped into each ear, and this may be repeated every four hours. Dry heat should also be applied by means of cotton wool and a rubber hot-water bottle to the side of the head.

Should suppuration occur the pain may be very severe and it can be relieved at once by paracentesis of the drum. As this is an extremely painful operation a general anæsthetic should be given. A timely paracentesis is believed by some pædiatricians to act beneficially in nutritional disease in infancy, and, apart from the relief of pain, the operation is desirable in very severe cases and especially in those occurring during scarlet fever and diphtheria. In others, spontaneous rupture may be allowed to occur, as it is found that this often gives a better result in the long run than an artificial opening.

The carbolic drops should be continued for a few days after the ear has begun to discharge. Syringing is not advisable at this stage. A fragment of dry, aseptic gauze should be left in the ear, and changed frequently if the discharge is profuse. The ear should be covered with cotton wool. This dry method of treatment may be continued until the discharge ceases; or syringing at intervals, varying according to the amount of the discharge, may be employed. Sterilised saline solution or boracic lotion should be used. The ear must be dried after the syringing and a plug of cotton wool placed in the ear. The treatment must continue *until the discharge has entirely ceased*. In most cases it lasts for two or three weeks. As soon as all swelling of the membrane has subsided, occasional inflation of the ear should be begun.

Should the discharge continue to be copious and the deafness marked, or if there is mastoid tenderness, the case should be referred to an aural surgeon without delay, as a mastoid operation may probably be required.

In children whose hearing is impaired owing to *accumulations of wax*, the deafness ceases rapidly when the wax is removed by syringing and the ears kept dry. *Enlarged tonsils* and *adenoids* should always be actively treated when the hearing is affected. *Chronic otorrhœa* is generally amenable to treatment, *provided* it is carried out thoroughly and persistently.

Among the poorer classes especially, however, the necessary thoroughness and persistence are often difficult to secure unless the treatment is undertaken by a properly trained nurse. The institution of regular aural school clinics, which began in Scotland some twenty years ago, has prevented a large amount of permanent deafness.

The ears must be regularly cleansed by syringing followed by drying and the blowing in of an unirritating antiseptic powder such as xeroform. This usually results in the diminution and gradual cessation of the discharge. In obstinate cases iodoform powder or boracic spirit may be substituted for the xeroform. In girls, if the head is dirty, the hair should be cut short. Swimming-baths and sea-bathing must be forbidden, and cotton wool should always be placed in the ears during washing of the head and neck.

If the middle-ear condition is complicated by eczema of the auricle, starch poultices followed by zinc ointment may be used; and if boils occur in the auditory canal they must be opened. Tough epithelial masses may be loosened by soaking with hydrogen peroxide.

Any signs of *mastoid disease* beginning should be watched for and when they appear the case should be transferred to an aural surgeon. Ophthalmoscopic examination is often a help in deciding if the mischief has extended beyond the middle ear; optic neuritis would point to involvement of the mastoid antrum.

CHAPTER VI

THE PHYSIOLOGY OF THE INFANT'S ALIMENTARY TRACT, AND SOME DERANGEMENTS OF ITS FUNCTIONS

Development of the Digestive Organs

Salivary Glands.—Even during the first week of life a little saliva is secreted which has some action on starch; but for the first six months the amount is so small as to be of no practical importance in normal digestion. During the second six months its quantity and its power of acting on starch steadily increase, so that by the end of the first year they are considerable.

Pancreas.—Before the child is born the pancreatic juice is found to act to some extent on proteins and neutral fats; and the intestinal secretion which stimulates the pancreatic ferments is also present long before birth. The pancreatic secretion is probably capable of acting slightly on starch from the first; but, like the saliva, it cannot be expected to do much in this way till after the sixth month.

Liver.—The liver is relatively large at birth and remains so during early childhood. "Well-formed bile-ducts are present in the twelve-week foetus, and there is bile-pigment in the intestinal contents at an even earlier date."¹

Stomach.—At birth the stomach is more tubular in form than later, owing to the slight degree to which the fundus is developed; and it retains for several weeks the nearly vertical position which characterises it during foetal life. The action of its muscular wall is relatively feeble.

During the first year the fundus increases in size more rapidly than the rest of the organ, and the stomach thus comes to lie even more horizontally than in the adult. The slight development of the fundus in young babies accounts for

¹ "Glandular Activity in the Human Foetus," M. F. Lucas Keene and Evelyn E. Hewer, *Lancet*, 19th June 1924, i., 111.

the small amount their stomachs are able to hold. According to Holt, the average capacity of the infant's stomach is at birth, $1\frac{1}{5}$ oz.; at one month, 2 oz.; at three months, $4\frac{1}{2}$ oz.; at five to six months, $5\frac{3}{4}$ oz.; and at twelve to fourteen months, nearly 9 oz.

Intestine.—In the young infant the intestine is relatively longer than in the adult. The muscular wall is also feebler, which helps to account for the greater tendency we find in little children to flatulent distension of the abdomen. At birth the cæcum is higher up, and more movable than in adult life, and the ascending colon is short. The main peculiarity of the intestine in the new-born infant is the large size of the sigmoid flexure, which at this age is nearly as long as all the rest of the large intestine put together. By the fourth month the other parts of the colon have grown so much that the sigmoid has nearly assumed its permanent proportion to them (Rotch).

Sucking and its Derangements.

The Act of Sucking.—In the adult there are two ways of drawing liquids into the mouth¹:—

(a) *Inspiratory sucking*, in which the suction power comes from the lungs, the buccal cavity being open behind for the time.

(b) *Mouth sucking*, during which the buccal cavity is closed behind and the active lowering or flattening of the tongue, which forms its floor, causes the indrawing of the liquid.

In the new-born child the process is one of mouth-sucking, but it differs a little from that in the adult. The infant's tongue, being as yet less mobile and more fixed in its position, as well as relatively larger, plays a less active part; and the lowering of the floor of the mouth is mainly due to vigorous downward movements of the lower jaw. As the child grows older (two to four years) he gradually takes to the adult way of using the tongue in sucking.

For the proper performance of the act of sucking the lips must be able to close firmly on the nipple, and the pharynx be shut off by the soft palate. The upper respiratory passages must also be free, so that breathing may go on with the mouth shut; and there must be no pain from the act. If

¹ L. Auerbach, *Arch. f. Anat. und Physiol.* (Physiol. Abth.), 1888, 59.

any of these conditions is not fulfilled, sucking may be greatly interfered with, and nutrition may consequently suffer. When a baby refuses to suck we should find out whether this is due to a disinclination for food, or is caused by some local interference with the act of sucking.

It is a striking fact that babies on the breast can breathe and take nourishment at the same time in a way that older children cannot do. This is due to the fact that in them the upper end of the larynx reaches higher and projects upwards behind the soft palate to such an extent that the milk and saliva readily pass round its sides and down the œsophagus without reaching the level of the glottic opening.¹

As the child grows older and his neck lengthens, the level of the glottic opening becomes lower in relation to the surrounding parts until at last it is below the uvula.

Interference with sucking may occasionally arise from malformation or disease of the parts involved in the act. Infants with hare-lip and cleft-palate, for example, have great difficulty in taking the breast or bottle at first; but with tact and perseverance some of them learn before very long to suck fairly well. It may be necessary, however, to resort to spoon-feeding or to the use of a syringe. Any painful condition of the lips, tongue, or palate stops a child sucking; and pain in the fauces often has the same effect. The baby may also refuse the breast or bottle if he has recent nasal obstruction or dyspnœa from pneumonia; for, in either of these cases, he has difficulty in keeping his mouth closed long enough for the process of sucking. Refusal to take the breast or bottle is, however, more frequently due to general causes, such as debility, dyspepsia, temporary interference with consciousness (as from injury at birth), or mental defect. When an infant is temporarily too nervous and excited to suck properly, small doses of chloral are sometimes useful.

Difficulty in Swallowing.—When a new-born child is unable to swallow more than two or three teaspoonfuls of fluid and almost immediately returns it, the case is probably one of congenital obliteration or stenosis of the œsophagus (p. 326).

Difficulty in swallowing is also met with in cardiospasm or so-called achalasia of the cardia. In this rare condition the

¹ L. Hasse, *Arch. f. Anat. und Physiol.* (Anat. Abth.), 1905, 328.

degree of obstruction varies greatly from time to time and is accompanied by an excessive flow of saliva and mucus (p. 334).

In addition to these rare and serious forms of difficulty in swallowing, a slight functional variety of the symptom is not uncommon in nervous babies who are being given semi-solid food for the first time. It is a purely neurotic symptom and is generally soon overcome by tact and perseverance on the part of the nurse.

Rumination (Merycism).

Much has been written on the practice of rumination in adults,¹ but the attention of the profession has only recently been directed to its practical importance as a habit in babies, and to the serious results it may have in them.²

As in the case of air-swallowing, the habit, or something like it, often occurs as a physiological act in normal infants. This we see mostly in strong, vigorous children who regularly take more milk from the breast or bottle than they require, and relieve themselves by regurgitating the surplus immediately after. The process, which is sometimes called *possetting*, has long been well known, and it has been recognised that exaggerated forms of it must not be mistaken for chronic vomiting. It is not usually a matter of any importance.

The *pathological* variety of the habit may develop at any age in connection with indigestion: but the most typical cases are those which begin in young babies without any obvious predisposing cause. It is one of the most characteristic "bad habits" of the early months of life. In some cases the habit is slight in degree, lasts only a short time, and does little harm; but in others it is severe and persistent and has a serious effect on the child's nutrition. The babies who develop it are generally of a noticeably neurotic type.

The symptoms are easily recognised when looked for. Shortly after a meal has been taken, the child assumes an air of abstraction, holds his head somewhat back, and makes chewing movements with his jaws—the mouth being wide open. A quantity of milk then wells up into the back of the throat. After gurgling there for a varying time, it may,

¹ E. M. Brockbank, *Brit. Med. Journ.*, 1907, i., 421.

² Freund, *Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, xi., 325; Grulee, *Amer. Journ. Dis. Child.*, 1917, xiv., 210.

if not large in amount, be swallowed again; but usually so much is brought up at a time that most of it runs out of the mouth and is lost. Consequently, though the child's appetite and digestion are normal, he becomes emaciated from lack of food. Before, and during, the regurgitation of the milk the infants often show obvious signs of gratification, and many assist the process by putting the fingers in the mouth. In most of the cases there are no symptoms of dyspepsia, and whatever food is retained is well digested. When the habit is given up the weight goes up normally at once; but so long as it continues the child remains thin, nervous, and restless.

The first important thing in the *treatment* of the condition is that the mother and nurse should be made to understand clearly that the whole trouble arises from what is merely a "bad habit," and that, if this is stopped, all will go well. The next point is that they must bring home to the infant—as they are usually able to do in a surprisingly short time—that the bringing up of the milk is a thing which incurs their displeasure and must not be continued.

It is a good thing if the baby can be induced to go to sleep at once after each meal; and, in the early stages of the treatment, the administration of small doses of bromide may sometimes be useful in helping him to do so. So long as he remains wakeful he must be watched and not left alone to practise his habit. On the slightest indication of its beginning, he should be taken up, talked to, and interested in some way or other. It may be necessary, however, to hinder the movements of the jaw by strapping up the chin. A more powerful deterrent is to introduce a piece of wood or bone like a horse's bit between the teeth before strapping up the jaw.

Children, who are old enough to sit up, generally do so before beginning to regurgitate the milk. Under these circumstances the child should be kept lying flat for some time after each meal; and, if he assists the return of the milk by putting his fingers into his mouth, this must of course be prevented—either by the use of elbow splints or in some other way.

If close and intelligent personal attention by the mother or nurse can be secured, the habit can usually be stopped in a short time. In three cases, in infants of four, five, and six months respectively, the habit, which was said to have begun

shortly after birth, had practically ceased, and a satisfactory gain in weight set in, within two or three weeks of the beginning of such treatment.

In those unfortunate babies whose mothers have not the time, or will not take the necessary trouble, to wean them from the habit, it is likely to persist for many months, and the danger of serious and sometimes fatal debility and emaciation is considerable. Grulee has found that a large proportion of these cases die.

Physiological Air-swallowing.

Under normal conditions, percussion and X-ray examination always reveal the presence of air in the stomach as well as in the bowel, and there can be no doubt that a moderate amount of it is a normal content of these viscera, and has a useful and necessary function there. Cannon has made observations which seem to show that the gas present in the stomach and bowel has an important action in keeping up their muscular tone. It was shown by von Mikulicz¹ that the thoracic portion of the œsophagus—between the level of the larynx and the cardia—also normally contains air.

There can be little doubt that the great bulk of the gas normally present in the stomach comes there by being swallowed. None of the natural processes which take place in the organ are capable of producing gas in any quantity; and when the gas is removed and analysed it is found to contain nothing but the ordinary constituents of atmospheric air, though these may be present in somewhat altered proportions. The source of the larger quantity of CO₂ usually found in it is due to the diffusion from the blood in the same way as occurs into the pulmonary alveoli.

According to Wyllie,² there are three ways in which atmospheric air may be taken into the stomach:—

(1) *Air-swallowing proper*.—In older children and adults the movements which take place in the mouth during deglutition cause a free admixture of air-bubbles with the food, saliva, and pharyngeal mucus, and a considerable amount of air is introduced into the stomach in this way. In infants, however, it

¹ *Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, xii., 569.

² "On Gastric Flatulence," *Edin. Hosp. Rep.*, 1895, iii., 21.

is probable that this form of air-swallowing occurs to a very small extent, if at all, because they have so much less saliva than older children and the movements of the tongue in them are so much less free.

(2) *Air-gulping*.—Under normal conditions, a certain amount of air is gulped down into the stomach during swallowing between mouthfuls of food and drink. This process takes place freely in babies. We are all familiar with the way careful mothers have of interrupting the infant's meal by placing him upright, and patting his back to help him to bring up again some of the wind he has taken down in this way. If something of this sort is not done, so much air may accumulate in the child's stomach as to lead to colic and vomiting.

(3) *Air-sucking*.—This method is similar to the air-swallowing which occurs in "crib-biting" horses. It differs from the other two in that it is not simply an act of swallowing, but depends, to begin with, on a *vis a fronte* exerted by respiratory movements. It is very doubtful whether air-sucking is ever practised voluntarily or instinctively in babies. It has been shown, however, that in them some air may be drawn into the stomach unintentionally during such spasmodic respiratory efforts as hiccough, sobbing and laughing, and during severe paroxysms of coughing.

Fate of the Air swallowed.—The natural sequence to the distension of the stomach with air in any way is the eructation of most of it, since only a small amount is required for physiological purposes. To enable this to take place freely in the case of a baby it is usually necessary, as we have already seen, that the child should be held upright occasionally, as he is unable to raise himself. In the case of older children, sitting up or running about is, in most cases, rapidly followed by the required eructation. The air which is not returned in this way gradually finds its way through the pylorus, and much of it is in time passed from the bowel.

Pathological Air-swallowing (Aerophagia).

In adults and older children abnormal aerophagia occurs frequently as a symptom of various forms of dyspepsia; and it is especially characteristic of neurasthenic and hysterical patients. Occasionally it is associated with rumination.

The air may be introduced into the stomach in any of the ways already mentioned:—

(1) *Air-swallowing proper*.—Although there is no reason to believe that this ever occurs to any extent in young babies, it occasionally develops to an excessive degree in adults and older children from their having got into the way of swallowing large quantities of air-containing saliva, in order to lessen the discomfort arising from undue acidity of the stomach contents.

(2) *Air-gulping*.—The abnormal form of air-gulping is not very uncommon at any age. In infants it is often associated with, and assisted by, the habit of sucking the fingers, a “comforter,” or some other object. When it is present as a “bad habit,” it has probably begun during some functional derangement of the digestion owing to its affording a degree of relief to the uncomfortable sensations arising from the condition of the stomach; but the habit, when once formed, tends to last long after the indigestion has ceased. In older children, as in neurotic adults, noisy eructations sometimes take place; these excite the interest of the patient’s friends, and this leads to his attention being increasingly concentrated on the habit and to its assuming an exaggerated form.

The symptoms of abnormal air-gulping in babies are often overlooked, owing largely to its being only practised when the child is left alone and no one is paying him any attention. In older children its occurrence is more easily recognised, being more noticeable from a distance. The child, if sitting, bends slightly forward; or, if lying, raises himself. He then shuts his mouth firmly, lowers his chin, assumes an air of absorption, and settles himself to swallow. As the air goes down, a slight clucking sound may be heard when it passes into the air-containing thoracic portion of the œsophagus. Then the mouth is usually opened and the wind comes up.

The process of air-gulping cannot go on if the mouth is kept open; and this gives an important indication for treatment. The ease and freedom of the process also depends largely on the amount of saliva present, as swallowing becomes difficult when the passages are dry. When the patient is left undisturbed the air-gulping goes on until he is tired, or until his supply of saliva is exhausted. While the air is being swallowed the up-and-down movements of the larynx are easily seen; and

on inspection and percussion of the abdomen, the stomach is found to be rapidly distending.

In some cases the air is quickly returned before it has passed beyond the œsophagus. In others it goes right down into the stomach. When this occurs, most of it is usually brought up again shortly, provided there is nothing to prevent the child sitting up; but, if he has to remain lying on his back or right side, the necessary eructations do not take place and the air gradually passes through the pylorus. Later, such of it as is not absorbed in the bowel is expelled from the anus.

In those cases in which the gastric flatus is readily regurgitated little inconvenience results from the habit; but when, for any reason, the wind is retained, unpleasant and sometimes serious results may follow. In many infants the passage of the wind through the bowel causes severe recurrent colic, along with restlessness and disturbed sleep. In a few instances the distension of the stomach, if extreme in degree and not soon relieved, may have grave consequences. It occasionally happens that weakly babies who are too young or too frail to sit up, and who have been left lying in their cots while their mothers were out or otherwise engaged, are found to have died suddenly; and, on post-mortem examination, nothing is found except extreme distension of the stomach and some collapse of the base of the left lung. Wyllie regarded it as probable that some at least of these cases were due to air-gulping, and one of us (J. T.) has seen several post-mortems which seemed to confirm his opinion.

In very rare instances, in older children, the patient is quite unable to return the wind from the stomach, even when he is running about. This may possibly be due to some slight anatomical peculiarity in the region of the cardiac orifice. It is probable, however, that it depends more, and perhaps altogether, on the special vigour and rapidity with which the stomach has been distended in these cases; it has been found that when the stomach is very quickly distended in adults by the use of effervescing powders, eructation is often impossible for some time after. Rapid and vigorous distension of the stomach from air-gulping gives rise to pain in the epigastrium, followed sometimes by severe cardiac and respiratory distress owing to pressure on the neighbouring parts, and, if its cause is not recognised, may occasion considerable anxiety.

Treatment.—In order to break the habit of air-gulping in infants, the mother or nurse must be on the look-out for the beginning of the swallowing movements, and, when any indication of them is noticed, must at once take measures to divert the baby's attention to other things. It is when he is feeling dull that he practises such habits, never when he is looking at things or being spoken to. If the child has the habit of sucking his fingers or any other object this must be stopped. Lastly, so long as the air-gulping continues, free eructation of the wind should be encouraged by giving a carminative from time to time, and by setting the child up and patting his back.

In older children it is important, to begin with, that the mother should explain to the patient that all the trouble is due merely to a bad habit which must be given up. If this is not sufficient, the next thing is to arrange for the insertion of a cork between the molar teeth whenever the first signs of air-gulping are noticed. This is a very effective measure. It not only stops the swallowing of air entirely for the time, but also helps greatly to discourage its recurrence, because it constitutes an annoying interruption to what the child has hitherto felt to be a soothing and restful habit.

(3) *Air-sucking.*—This is said to occur occasionally in older children, but probably never does so in babies.

Appetite and Thirst.

It is often difficult to distinguish hunger and thirst in babies, and we must remember that an infant's desire to suck is oftener due to thirst than to hunger. When we see a baby opening and shutting his parched mouth and straining wistfully after any bottle or cup that is brought near him, it is boiled water, not milk, that should be given. If for any reason fluids cannot be given by mouth, a saline enema (2 oz.) should be administered; and, if that is not retained, subcutaneous, intravenous, or intraperitoneal saline infusions are indicated. An abnormally large appetite in a baby is often merely a bad habit induced by habitual over-feeding.

When an infant refuses the bottle, and there is no reason to think that sucking hurts him, the cause is usually to be found in dyspepsia or improper feeding. Disinclination for

food is the natural accompaniment of a diminished power of digestion, and with recovery of the digestive power the appetite returns. An excessive amount of cream in the milk often causes it. Obstinate refusal to suck is sometimes a striking symptom in the early stages of pyloric stenosis, when the food is accumulating in the stomach and the child has not yet learned to relieve himself by vomiting.

Normal Digestion.

As we have seen (p. 119), the anatomical capacity of the young infant's stomach is small. There is, however, reason to believe that its physiological capacity, so to speak, is much greater than post-mortem measurements had led us to believe, and that it is neither necessary nor desirable to limit the size of the meal strictly to the quantity that the stomach can hold at one time; for X-ray examinations show that a considerable amount of the milk passes into the duodenum during the nursing. This is interesting in relation to the fact that some modern pædiatricians do not limit the amount of the feed, but permit the child to satisfy his appetite.

After the milk enters the stomach the bulk of its protein is coagulated by the action of the rennet. The curd produced in this way contains the casein and some of the fat, along with calcium and other salts from the whey; while the whey itself contains the remaining constituents of the milk which are soluble in water (salts, sugar, albumin, and globulin). The whey soon passes into the duodenum; but the curd, if considerable in amount, remains much longer in the stomach. There has been great difference of opinion as to the time it takes for the healthy infant's stomach to empty itself after a meal. According to Morse and Talbot, "the stomach digestion lasts in the breast-fed baby from one and a half to two hours; and in the artificially-fed baby, three hours"; but it has also been shown that in some breast-fed infants the stomach may be empty within an hour. The duration of digestion depends, of course, to some extent on the quantity of food taken.

The opening of the pylorus to let milk into the duodenum depends on the reaction of the stomach contents and on that in the duodenum. Acidity of the pyloric end of the stomach

predisposes to the pylorus opening, and strong alkalinity to its closure; while alkalinity in the duodenum tends to make the pylorus open and acidity to keep it shut. Should the contents of the pyloric portion of the stomach be strongly acid, therefore, their passage into the duodenum rapidly inhibits the opening of the pylorus. The cardiac orifice relaxes when the contents of the adjoining portion of the stomach are alkaline, and closes when they are acid. It is probable that solids, such as coagulated casein, have a mechanical action in preventing the opening of the pylorus.

Protein.—The protein of the food must be completely disintegrated into the various amino-acids of which it is composed before it can be rendered suitable for absorption and assimilation. The first step in the process takes place in the stomach, when the casein is coagulated, and it and the lactalbumin are rendered soluble by their conversion into albumoses and peptones. It is, however, in the intestine, by means of the activity of the succus entericus, that the complete breaking down into the amino-acids takes place. Most of the protein is absorbed and that which is not utilised is excreted in the urine, for the most part as urea. The fæces do contain a certain amount of protein, but it is impossible to state how much of this results from the food ingested and how much from the secretions and the normal bacterial inhabitants, unless in the case of casein curds which tend to occur when the child is fed with unboiled cow's milk. Protein in the bowel, in contrast to carbohydrate, exerts an anti-fermentative effect and thus tends to constipation. This action is seen in feeding with undiluted cow's milk, and is taken advantage of by Finkelstein and Meyer in the preparation of their *eirweiss milch*.

After absorption the protein serves three purposes. In the first place, like the fat and carbohydrate, it acts as a source of heat and energy. It must, however, also repair the wear and tear of the tissues, and in addition, what is particularly important, provide for the formation of new tissues in the growing infant and child. The first call on the protein is for the supply of energy, then the repair of waste and finally growth are provided for. Thus in inanition, so long as sufficient for the first two functions is supplied, there may be no loss of weight, but if ample for these two processes is not forth-

coming loss of weight is inevitable. Protein, it must also be remembered, possesses a peculiar specific dynamic action in that it stimulates metabolic activity and therefore calls for increased nourishment. This is a point of importance in the dieting of a condition such as diabetes, where the tissues can only deal with a certain much diminished maximum.

It is important to bear in mind that, so far as the wear and tear and growth are concerned, protein is only of value in virtue of the particular amino-acids of which it is composed, and that different proteins contain the various amino-acids in different quantitative and qualitative combinations. Cow-milk protein, for example, contains much less leucin, lysin, and the absolutely essential tryptophan, than does the protein of human milk. Now, as these amino-acids are resynthesised to form the protein of the body, a much larger amount of cow-milk protein is required in order that the requisite quantity of these necessary elements will be forthcoming. Hence it is quite apparent that the different proteins are not quantitatively replaceable. This is a matter of fundamental importance in the substitute feeding of the infant.

Fat.—Fat, and especially animal fat, is an essential constituent of the diet of the child and the infant. This is not because of the fat as such, since the body has the power of manufacturing it from carbohydrate, but because of the growth-promoting factor, vitamin A, which is essential for growth and is always associated with the fat. Fat is, however, a valuable form of food because it is the most concentrated source of energy and animal heat. It is generally admitted that the infant has more need of heat producers than the adult, because the body surface is relatively larger and his loss of heat correspondingly greater. It may be that in this way we can explain the abundant adipose covering with which he is naturally supplied. It is not improbable, too, that this is the reason that the natural food of the infant is relatively so rich in fat. Instead of amounting to only $\frac{1}{7}$ of the proximate principles, as is recommended in general dietetics, fat amounts to at least $\frac{1}{4}$ of the proximate principles. The proportion of the fat is even greater in the case of cow's milk, a fact which may be explained by the more rapid growth of the calf, and hence a greater need for vitamin A. Fat is also necessary for the health of the nervous system, and, as it assists in the

absorption of calcium and phosphorus, and other salts from the bowel, it is specially important for the growth of the bones and teeth.

The digestion of fat begins in the stomach, though most of it takes place in the bowel as a result of the action of the pancreatic juice. Neutral fat as such cannot be absorbed from the bowel and must be split into fatty acids; hence in disease of the pancreas the fæces contain an excessive amount of neutral fat, which may be so abundant that the motions are as if bathed in oil. Normally, more than 95 per cent. of the fat is digested and is present in the intestinal contents as free fatty acid or combined with calcium or other salts in the form of insoluble soaps. The relative proportion present as fatty acid and soap is influenced by the amount of available salts and especially calcium. An excessive amount of calcium will tend to increased soap formation, which will interfere with the absorption of the fat and calcium. This increased production of insoluble soaps causes an increased fæcal output, a feature observed in all diets with a high calcium content, *e.g.* undiluted cow's milk. Since phosphorus is only utilised in equal amount to that of calcium, deficient absorption of the latter leads to deficient retention of phosphorus. The phosphorus, unlike the calcium, may be absorbed, but the excess over that required to combine with calcium is excreted by the urine. This interdependence of calcium, fat, and phosphorus absorption and utilisation is one of the most important fundamental features of infant metabolism.

The absorption of fat even after satisfactory splitting may also be interfered with when there is a deficiency of bile, and again the utilisation of calcium and phosphorus will be affected. This is observed in congenital atresia of the bile ducts and it may be mentioned that a deficiency of bile salts may play a part in the pathogenesis of coeliac disease.

Normally, the proportion of the fat in the diet which is absorbed is very high—over 90 per cent., although it varies somewhat according to the type of fat given or the amount ingested. It is stated that the percentage absorption is better when human milk is the source than when the diet consists of cow's milk. This relatively better absorption with the natural food may be taken as the general rule with regard to all the constituents. When insufficient amounts are given the

proportion absorbed is less than when the child's needs are fully supplied. Hence, before any conclusions can be drawn regarding the question of the proportion absorbed, the normal quota for the particular age of the child must be supplied. In health, the greater amount given the higher the percentage absorption. In disease, *e.g.* coeliac disease, the reverse is the case. This better absorption with the larger quantities of fat probably depends on the fact that so much is required in the bowel for lubricating purposes, and it would seem that its provision is the first call on the amount supplied.

The fat which is not absorbed is excreted in the fæces and, whatever the quantity ingested, fat or its derivatives rarely form less than 30 per cent. of the total fæcal output. An excessive amount of fat excreted tends rather to a greater bulk of fæces than to a greater proportion of fat in the fæces, though with looseness of the motions the fat may come to form as much as 60 per cent. One point cannot be too strongly insisted upon, *viz.*, that the fat content of the fæces does not necessarily bear any relationship to the proportion of the intake which is absorbed.

Fat is undoubtedly the most difficult proximate principle of the food for the child to digest. A form of dyspepsia (fat dyspepsia—"bilanzstörung" of Finkelstein), characterised chiefly by large soapy stools and a failure to gain weight, results if a milk too rich in cream is supplied, or even in some children when the fat is in the normal proportion. In this connection one thinks of the success in infant feeding obtained by the Belgians with butter-milk. From what we have already said regarding the interdependence of the metabolism of fat and the minerals, this response to a too rich fat diet is easily understood. It is probably for this reason that butter-milk (*i.e.* milk from which the butter had been removed) was such a favourite infant food with the Belgians, and it is for this reason that the makers of dried milk, *e.g.* "Glaxo" and "Cow and Gate" varieties, now supply a form which contains only one-half of the normal amount of cream. It should always be borne in mind that fat is not well tolerated in the presence of acute and chronic disease of the alimentary tract, and the same is true when children are fevered or during very warm weather.

Milk-sugar or *lactose*, the formation of which is a special function of the mammary gland, is the form of carbohydrate which is present in the milk of all mammals, the only difference

being in the quantity present. Lactose is a di-saccharide, being formed from the combination of glucose and galactose. It is digested in the small intestine when it is converted into the monosaccharides dextrose and galactose, and as such is absorbed into the blood stream. Some of it may be burned directly, but the bulk is transported to the liver and muscles and deposited as glycogen. It is because glycogen combines with so much water, two or three times its own bulk, that a carbohydrate-rich diet causes a retention of fluid and increase in weight. From the liver and the muscles the glycogen is gradually doled out to the blood as required. This so-called mobilisation of the carbohydrate is under the influence of the ductless glands, especially the adrenals and thyroid, and for its combustion insulin, the internal secretion of the pancreas, is necessary.

Sugar is an important and ready source of muscular energy and heat. It is for this reason that intravenous injections of glucose are so widely practised in the infant. It spares the protein and from it fat can be formed. It is also necessary for the complete combustion of fat. As Rosenfeld says, "In order that complete combustion of fat may take place an equal quantity of carbohydrate is required." When a less proportion of carbohydrate is available there is incomplete combustion of the fat, with the formation of acetone and beta-oxybutyric acid and the development of ketosis (p. 551).

Sugar, like protein, is essential for life. Protein is in part burned as carbohydrate, and, if there is an insufficient amount in the diet, or available from the protein in the food, the proteins of the tissues are utilised and rapid wasting results.

Carbohydrate, and especially sugar, from its fermentation in the gut, exerts a laxative action and, when given in a larger proportion than 10 per cent., is apt to cause diarrhoea, with loose, green and acid stools.

The *mineral salts* (P, Ca, Mg, Fe, Na, and K) are, like the protein and carbohydrate, absolutely necessary for life, since they are essential constituents of the tissues. Their great importance in nutrition has within recent years been stressed by Orr of the Rowatt Institute, who would place them in importance along with the vitamins. So far as the infant and growing child are concerned they have, like the protein, not only to provide for the various physiological processes as well as for wear and tear,

but also for the growth of new cells, the skeleton, and the increased formation of blood.

The salts are present in the food in the form of insoluble compounds and before absorption must be rendered soluble. This is brought about by the action of the hydrochloric acid of the gastric juice. In this soluble state they enter the duodenum and are absorbed so long as the reaction remains on the acid side of neutrality. Telfer has found from experiments in animals that a feebly acid reaction of the intestinal contents continues to the middle of the ileum, so that it would seem justifiable to conclude that a certain proportion will remain in a soluble form and be capable of absorption to this level.

It will thus be understood how anything which increases the acidity of the intestinal contents—fermentation or the administration of an acid—will favour their absorption, and factors which render the reaction alkaline will have the opposite effect. As previously mentioned, the amount of fat present in the gut and its absorption also affect the absorption of the salts. An excessive amount of fatty acids, by fixing the lime and forming an insoluble soap, definitely interferes with the absorption of this element. The relative proportion of the various salts themselves may also interfere with normal absorption. An excess of lime over the phosphorus may fix an excessive amount of the latter by forming insoluble calcium phosphate and interfere with its absorption. And finally, within certain limits, the amount absorbed is directly related to the amount ingested. With milk feeding this is a point of no significance so far as calcium and phosphorus are concerned, but it is quite different in the case of iron, the content of which in milk is exceedingly low and barely sufficient to supply the infant's needs. Thus, when milk is diluted, anæmia may ensue from iron starvation.

The salts which are not absorbed are passed in the fæces and the quantity of this residue, like that of the fat, although these two quotients are intimately related, influences the bulk of the fæcal matter. The salts which are not utilised after absorption are for the most part excreted in the urine. There is no doubt, of course, that salts can be excreted by the gut, since the intravenous injection may cause more to appear in the fæces than was ingested in the food, but normally this route of salt excretion is probably of little importance.

The *water* in the food is of no less importance than the other constituents. When we consider that the tissues of the infant's body contain 66 to 69 per cent. of water, and appreciate that the body-weight is trebled during the first year of life, it is not surprising that Nature has supplied him with a very water-rich diet. Human milk contains 88.5 per cent. and cow's milk 87.4 per cent. of water. It is estimated that the child requires per day 105 grammes of water per kilo of body-weight as against only 40 grammes required by the adult.

Although the greatest proportion of water is obtained from the food, it must be remembered that a certain quota is derived from the combustion of the hydrogen of the foodstuffs. We have also seen that excessive carbohydrate feeding causes retention of fluid. This fact makes the balancing of the water intake with the water loss exceedingly complicated and difficult of estimation. The water is absorbed from the gut and leaves the body *via* the kidneys, lungs, skin, and intestine.

Water plays a most important rôle in metabolism. It enters into the formation of all the tissues; it is essential for the keeping in solution of many active ingredients; it takes part in many reactions and in the processes of anabolism and katabolism; it acts as the means of transport of products involved in the above to and from the tissues; it is one of the great adjuncts to excretion, and in consequence of evaporation from the lungs and skin is an important factor in the regulation of the temperature of the body.

It is the variation in the water content of the tissues which is chiefly responsible for rapid fluctuations in weight. This, of course, is related to the salt and glycogen contents of the tissues. An increase in the absorption and retention of salts, as may follow the institution of a high salt diet, brings about an increased retention of fluid and a rise in weight. This is not infrequently observed when the child's diet is changed from human milk to cow's milk, *i.e.* from a diet containing 0.2 per cent. of salts to one containing 0.7 per cent. of salts. This is a point which should always be considered in evaluating the result from a change of diet, and similarly a rich carbohydrate diet, by increasing the glycogen content of the tissues, also leads to a retention of fluid. On the other hand, an excessive loss of salts from the tissues, as occurs from the gut in enteritis, leads to rapid and great loss of weight.

Reference has already been made to vitamin A—the growth-promoting factor. This is, however, only one of the four recognised accessory food factors. There are in addition the anti-neuritic substance or vitamin B, the anti-scorbutic factor or vitamin C, and the so-called anti-rachitic factor or vitamin D. With the exception of vitamin C these substances are apparently very stable. Vitamin C or the anti-scorbutic factor is, however, comparatively unstable; it not only disappears from the milk on standing, but it can be destroyed by prolonged heat and would appear to suffer in activity even from undue handling of the milk.

Defæcation and its Disorders.

The infant's bowels generally act from two to four times in the day during the first month or two, and once or twice daily after that. The process should, of course, be quite easy and painless. If otherwise, the cause of the discomfort or pain requires investigation.

Scanty and infrequent motions may be due simply to the small amount of food reaching the intestine, as occurs when there is persistent vomiting, or to a variety of other causes (see Constipation, p. 152). Painful defæcation may be due to fissure or other cause of spasm of the rectum or to tenesmus.

Fissure of the Rectum and Rectal Spasm.¹—Anal fissures are by no means rare even in early infancy, and their occurrence must not be forgotten, for they may give rise to severe and perplexing symptoms. The lesion, if single, is most frequently situated on the posterior wall. In some cases it is easily seen by merely pulling apart the edges of the anal orifice. In others a small speculum has to be used. In babies a bivalve nasal speculum answers the purpose. Should the rectal mucous membrane be partly prolapsed, as sometimes happens, the fissure may be difficult to find.

Causation.—Two factors are to be recognised in the causation of fissures. On the one hand, there is local weakness due to lesions of the skin and mucous membrane, especially eczema, specific eruptions, or an irritation arising from diarrhœa or thread-worms. The habitual use of suppositories or enemata is apt to cause them. On the other hand, there is the periodic

¹ F. Frühwald, *Ueber Mastdarm-Rhagaden und Fissuren in Kindesalter*, Wien, 1896.

over-distension of the anal ring by hard fæcal masses, which takes place in constipation.

Symptoms.—The first effect of the fissure is spasm of the sphincter ani. This may be brought on only by movement of the bowels, or it may occur at other times also. When the attack comes on, the infant screams and stretches himself straight out with his head back and his thighs extended and pressed closely together, and he seems in severe pain until the spasm passes off. The occurrence of rectal spasm tends greatly to increase the constipation. Defæcation becomes extremely painful and the infant does all he can to postpone it. The longer the motion is retained the larger and harder does it become, and the more painful is its ultimate passage, so that a vicious circle is set up. The motions are often streaked with blood. The frequently recurring, vague, sickening pain which the child suffers soon has a marked effect on his appearance. He becomes haggard, worn, restless, and miserable. The screaming which rectal spasm causes is often mistaken for colic; but, if the child's attitude and behaviour during the attack are watched, there should be no difficulty in recognising the nature of the case.

In some cases the irritation spreads to the nerves of neighbouring parts. The surrounding skin may become tender to the touch, and there may be erections of the penis or spasmodic retention of urine. This last-named symptom should always draw attention to the state of the anal orifice. General irritability, sleeplessness, prolonged and repeated screaming, and even convulsions, may owe their origin to this small lesion.

Treatment.—The attacks of rectal spasm are generally much relieved by the application of heat to the perineum either in the form of a hot bottle or sponge, or a hot hip-bath.

In the treatment of the fissure the first object to be aimed at is the removal of the constipation by diet, massage, and gentle laxative medicines. If the motions are always kept soft, the parts round the anus clean, and the orifice anointed with a suitable ointment, recovery usually takes place in a short time. An ointment containing ichthyol and tannic acid—a drachm of each to the ounce of vaseline—will be found useful. It should be gently insinuated into the anus on a firmly rolled-up pledget of cotton wool. Should the anal spasms continue, the anus should

be stretched with the finger, under an anæsthetic. This simple proceeding is generally entirely successful.

In any case of severe and recurrent rectal spasm in which no fissure or other local cause is found, stretching of the sphincter should always be tried. It often leads to complete and lasting relief.

Tenesmus.—Straining at stool is a common symptom in infancy. It occurs in most cases where the lower end of the bowel is irritated either by a primary local disease (*dysentery*) or by the passage downwards of abnormal fæces. It may also be due to lesions higher up; it is often, for example, an important symptom of intussusception as well as of ordinary prolapse. It may also be due, in older children, to a stone in the bladder or to severe phimosis. The condition, when acute, causes much distress. When chronic, its presence may only be indicated by the child's disinclination to be removed from the chamber after the motion is passed. The symptom generally signifies the presence of a condition which will be benefited by copious irrigations of warm water. If it is severe, a small starch enema, containing a few drops of laudanum, may be given.

Rectal Prolapse.—It should be appreciated that the rectal mucous membrane always protrudes to a certain extent during defæcation; immediately after the act, however, the prolapsed mucous membrane returns spontaneously. Parents are apt to mistake this for true prolapse. In true prolapsus ani the whole thickness of the wall of the rectum is involved (Fig. 64), so that the protruding bowel corresponds to the invaginated portion of an intussusception. In these cases the prolapse may be from two to nearly six inches long, and it may remain constantly down.

Causation.—The greater tendency to prolapse which obtains in early life is attributed to many things. For example, the sacrum is less curved in children, the rectum straighter, and the muscles and interstitial tissues in the pelvis weaker and more yielding. The rapid wasting which so readily occurs in infancy is a strong predisposing cause of prolapse. It lessens the support afforded to the rectum by the ischio-rectal fat before the muscles have time to accommodate themselves to the greater strain thus thrown upon them. The exciting cause is severe and long-continued straining. This may result from constipation, from thread-worms, or from diarrhœa associated with

catarrh of the rectum. Extreme phimosis may also lead to it, and in older boys it is sometimes a symptom of vesical calculus.

Symptoms.—When slight in degree the prolapse only occurs during defæcation, and is readily returned. In severe cases, however, any exertion, such as crying and coughing, brings it down, and it is more difficult to return satisfactorily. When the bowel remains long down it is apt to become inflamed and ulcerated.

Diagnosis.—There is usually little or no difficulty in recognis-

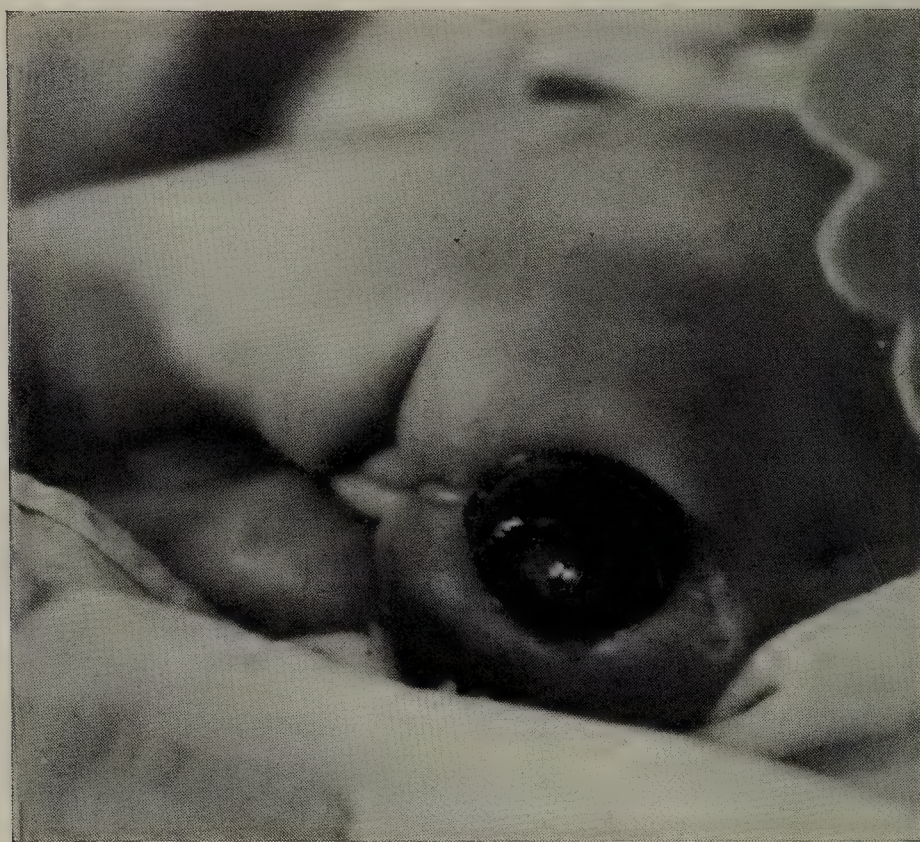


FIG. 64.—Rectal Prolapse in a child of 3 months.

ing the nature of a rectal prolapse. Digital examination reveals that the mucous membrane is reflected at the anal ring and that the finger cannot be inserted into the rectum around the tumour as in intussusception. Occasionally a true intussusception is protruded from the anus. Although intussusception should be diagnosed long before this happens, the above-mentioned digital examination and the other signs (abdominal tumour) and symptoms make the differentiation easy.

Treatment.—In the milder degrees it may be sufficient, after washing the prolapsed bowel with cold water, to reduce it by gentle pressure and by strapping the buttocks together for several days to effect a cure. At the same time attention should

be paid to any diarrhœa or constipation or any other factor which leads to straining.

If, however, the condition is severe and of any duration and accompanied by diarrhœa, then more heroic measures are necessary. A very successful method,¹ which one of us (L. F.) learned from Professor A. d'Espine of Geneva, is the injection of absolute alcohol into the perirectal tissues. It is possible that the alcohol sets up an irritation and causes by adhesions a fixation of the rectum to the surrounding tissues. In order to avoid all straining the operation must be performed under full anæsthesia, and it is advisable to empty the lower bowel by means of an enema. The prolapse, if down, is reduced and the perineum washed and sterilised with iodine. With the finger in the rectum to act as a guide, 1.5 c.cm. of absolute alcohol are then introduced on each side into the perirectal tissue at a depth of 2 in. to 2½ in. An ordinary exploring syringe is employed, the needle being inserted on each side about ¼ in. from the anal margin. The needle punctures are sealed with collodion, a pad placed in the perineum and kept in position by strapping the buttocks firmly together. Instructions are given that the child must only move the bowels while in the recumbent position, the fæcal matter escaping by the side of the dressing. The pad and strapping are reapplied daily for a week; by this time it has been found, as a rule, quite safe to discard all appliances. In 37 out of 41 cases (90 per cent.) of prolapse, of all degrees of severity, a complete cure was obtained. Occasionally the operation has to be repeated once and it may be twice: this was so in three cases which were ultimately cured. As a rule, however, once is sufficient. There would seem to be no danger connected with this procedure, as untoward after-effects have never been observed.

The Fæces.

The **meconium** passed during the first two to five days is of a dark greenish-brown colour, of a viscid semi-solid consistence, slightly acid in reaction, and without odour. At birth it is sterile; but, within from eighteen to twenty-four hours, micro-organisms find their way into it, at first through the anus and later through the mouth.

¹ *Brit. Journ. Child. Dis.*, April to June 1921, xviii., 83; also L. Findlay and J. B. D. Galbraith, *Lancet*, 13th January 1923, i., 76.

It contains epithelial cells and mucus from the intestine and its colour is due to bile, but the bulk of it is composed of matter derived from the vernix caseosa which the infant has swallowed along with liquor amnii during intra-uterine life. That the child does swallow liquor amnii is proved by the fact that meconium always contains hairs and squamous epithelial cells which must have come from the surface of his body.

The motions lose the character of meconium usually after three or four days and become, in turn, dark green, yellowish-green, greenish-yellow, and at last the normal orange-yellow colour. They also become more fluid for a time and then gradually regain their semi-solid consistence. The exact time and extent of these changes vary considerably within physiological limits; and, as Southworth has pointed out,¹ much importance need not be attached to a mere atypical colour or consistence of the stools, so long as the baby seems well and is gaining satisfactorily in weight.

Normal Fæces.

In the healthy breast-fed infant the motions have an orange-yellow colour and a uniform semi-solid consistence; the reaction is slightly acid and they have a somewhat sour but not offensive odour. The stools of a hand-fed baby are similar, provided the food he is taking resembles breast-milk in composition and is being well digested.

Their Composition.—The infant's fæces consist of those foodstuffs which have not been absorbed, of various secondary products of digestion, and of bacteria.

(a) *Unabsorbed Foodstuffs.* — A considerable proportion of the stool is composed of fat, roughly 30 per cent.² Normally there is very little neutral fat present, the bulk of the fat being present in the form of soaps or fatty acids, the relative proportions of these depending on the amount and relative proportions of the various salts. An excessive amount of calcium in the fæces causes a relative increase in soap formation: this occurs in rickets. It is the excessive amount of salts, especially of calcium and magnesium, which give the motion of the bottle-fed child its bulky, pale, and dry appearance from the excessive amount of soap formation. The splitting of the fat is caused by

¹ "The Stools of the New-born," *Arch. of Ped.*, 1910, xxvii., 167.

² H. S. Hutchison, *Quart. Journ. Med.*, 1920, xiii., 277.

pancreatic secretion. Neutral fat is not absorbed and hence appears in the motion, and when present in appreciable amount as an oily substance should raise the question of disease of the pancreas (pp. 144 and 524). One of the chief aids to absorption of fat is bile, and hence in jaundice due to biliary obstruction an excessive amount of fat is present, but either as free fatty acid or soap.

Protein from the food enters very little into the protein-content of the fæces. The only exception is when unboiled cow's milk is given and large casein curds appear.

No carbohydrate appears in the stools unless an excess of starch is being given.

(b) *Secondary products of digestion.*—Certain alkaline products are formed by putrefactive bacteria from unabsorbed proteins higher in the intestine. Others, which are acid, arise from fermentation of carbohydrates—either starch or excess of sugar, or a normal amount of sugar in a damaged intestine. These products of fermentation are lessened by the putrefaction and alkali-formation which follow an increase of the protein in the food.

(c) *Bacteria.*—As much as 16 to 18 per cent. of the fæces may be formed by micro-organisms—many of them probably dead. Under normal circumstances, living bacteria swarm in the lower bowel, while the upper part of the small intestine is sterile. If the mucous membrane of the small bowel has been damaged, however, either by a too concentrated solution of whey salts or otherwise, the bacteria of the large intestine may spread upwards and flourish more or less in the ileum.

In **starvation**, the scanty stools have a dark brownish-green colour like that of meconium, and a slightly stale odour. They may be constipated or loose. They are mainly composed of bile, intestinal secretions, and bacteria, and they may contain bile-stained mucus. Motions with these characters, the so-called *hunger stools*, must not be mistaken for evidence of enteritis. They are met with in severe cases of congenital pyloric stenosis when little or no food is getting beyond the stomach.

Abnormal Constituents in the Stools.

Under various conditions we meet with abnormal substances in the infant's motions, such as casein or fat curds, vegetable tissues, masses of undigested food of other kinds, intestinal

sand, medicinal substances, mucus, pus, blood, membranous fragments, and parasites and their ova.

Curds are often found in the stools, and casein curds are to be distinguished from those formed mainly of fat. The *casein curds* are often large, and they are of a white or yellow colour. They are hard and tough, and sink in water; and are rendered much harder by the addition of formalin; they are insoluble in ether. The *fat curds* are white, yellow, or greenish and small in size. They are readily broken up by pressure and float in water. When acidified and heated they dissolve in ether, and they are not changed in consistence by the action of formalin.

Liquid fat is found in the motions in certain examples of disease of the pancreas and in that rare congenital and occasionally familial error of metabolism which is known as '*steatorrhœa*.'¹ In the latter condition, when the motions cool, the oily material solidifies into a crust or cake of solid fat, which has earned for the excreta the name of "butter-stools." Seldom, even when the fat-splitting is more defective and the proportion of fæcal fat greater, apart from congenital steatorrhœa, has the motion such a definitely fatty appearance. So striking, indeed, is this appearance that mothers have complained that the child was passing grease. It is interesting to note, too, that although the percentage absorption of the intake of fat is no higher than that which occurs in a severe example of coeliac disease (p. 305) and that the protein, as well as the calcium and phosphorus of the diet, are poorly utilised, these children do not suffer in their nutrition. In the case of congenital steatorrhœa, however, there is no evidence of a disturbance in the carbohydrate metabolism, whereas in coeliac disease this is usually present. The same is true of those cases of organic disease of the pancreas which develop in later life and are accompanied by wasting. It has been surmised that the condition of "congenital steatorrhœa" is due to some anomaly of the pancreas, but tests carried out in the few cases observed have failed to disclose any definite defect in the secretion of the gland.

Fragments of vegetable tissues, such as fibres of banana and gritty particles from pears, are occasionally found in the motions of older infants who are injudiciously fed. The former

¹ A. E. Garrod and W. H. Hartley, *Quart. Journ. Med.*, 1912-13, vi., 242; R. Miller and H. Parkins, *ibid.*, 1920-21, xiv., 1; A. Sœcknick and F. Thoenes, *Jahrbuch. f. Kinderheil.*, 1927, cxv., 315.

are sometimes taken for worms. Pear grit must not be mistaken for **intestinal sand**,¹ which is a rare constituent of children's stools, and consists in small semi-transparent, gravel-like fragments composed mainly of calcium phosphate. I have found it in the motions of a baby as young as nine months. The causation of the condition is unknown.

Some **medicines**, such as iron, bismuth, cod-liver oil, and petroleum, can often be recognised in the stools.

Mucus is present in small amount in most normal, and in large quantity in many abnormal motions. When it is present in sufficient amount to be of clinical importance it can usually be recognised by the naked eye, though undigested starch may sometimes have a somewhat similar appearance. Its presence may be due to irritation of any part of the intestine, not only in consequence of disease, but as a result of purgation with castor oil and lavage of the bowel with saline. It is important to remember these latter causes and not ascribe the presence of mucus in the stool to disease without due consideration.

When it comes from the small intestine the mucus is thoroughly mixed with the fæces, while that from the lower bowel is more obvious, being more on the surface of the stool.

Pus may be found in considerable quantity in the motions during the later stages of severe colitis and in ulceration of the large intestine.

Blood.—In infants the presence of small quantities of blood in the stools is often of little importance. Streaks of it may be seen in the motions in various forms of diarrhœa, and often also in constipation, especially if there is any ulceration, fissure, or prolapse of the rectum present. Blood and mucus in moderate amount and passed alone with straining is very characteristic of ileo-colitis. In large amount they form the classical, and at times, it is important to remember, possibly the only symptom of intussusception.

Hæmorrhage from the bowel occurs in ulcer of the duodenum and in other intestinal lesions; and it is a frequent symptom in the hæmorrhagic disease of new-born children (*melæna neonatorum*, p. 493). *Spurious melæna*—that is to say, the passage from the bowel of blood which has been sucked from fissures in the mother's nipple or swallowed during an attack of epistaxis—is far commoner than true *melæna*.

¹ A. E. Garrod, *Trans. Med. Chir. Soc. Lond.*, 1901, lxxxiv., 389.

Fragments of **membrane** may be met with in infancy from severe colitis, and sometimes from other causes.

Worms and their ova are often present in the stools; and, in rare instances in hot weather, the larvæ of certain flies may be passed from the bowel.

Other Characters of the Motions.

The *number* and *size* of the stools vary in health as well as in disease. It is always more important to ascertain the amount of fæces passed in the twenty-four hours than the number of the movements; for the latter often depends largely on the state of the child's natural disposition. A restless, lively baby may have three or four normal motions in the day, while one who is placid and contented passes the same amount in one large daily stool.

If the stools are very large, this usually implies a diminution in the absorption of nourishment from the bowel, and is therefore apt to be accompanied by a serious failure of strength. It occurs chiefly when the digestion in the small intestine is interfered with. When, in a case of diarrhœa, the motions, though frequent, are small in size—as is the case when the lower bowel is the part chiefly involved—the interference with the child's nutrition may be comparatively slight.

The *colour* of the milk fæces, which is normally bright yellow, varies considerably in shade and depth within physiological limits, the exact tint depending on the amount of bilirubin present. It is lighter when the digestion is taking place slowly, consequently the stools of hand-fed babies are often a paler yellow than those of infants who are on the breast.

The colour is generally altered in disease and a change to some shade of green is very common. This is generally of little or no importance; normal stools often turn green on standing, and this by crushing the stool will be seen to affect only the surface. Green motions may be due to strong acidity or to alkalinity, to the presence of a special ferment or to *Bacillus pyocyaneus*. The addition of nitric acid to a green stool decolorises it if the abnormal tint is due to micro-organisms, and gives a play of colours if it is caused by biliverdin. In starvation, as already mentioned, the motions

have usually a dark brownish-green colour. The addition of malt or barley-water to food may also cause a slightly brownish tint.

Clay-coloured motions often arise from obstruction of the bile-ducts, but they may also be due to a colourless bile-salt having replaced the usual coloured one. A chemical examination is necessary to determine whether bile is present. As previously mentioned, the presence of a large proportion of soap in the motions may also make them clay-coloured or white, as well as hard and dry. When a newly-born child's stools are white from the first, this usually depends on congenital obliteration of the bile-ducts, but it may also be caused by obliteration or obstruction of the bowel below the point of their entrance.

The administration of bismuth, iron, and charcoal blackens the stools. The darkest motions, however, are those which contain altered blood.

The soft homogeneous *consistence* of properly digested milk-fæces is lost when the child's food is varied, and is more or less changed in most forms of dyspepsia. The presence of much soapy matter renders them dry and hard, while an excess of sugar or cream usually tends to make them too liquid. In summer diarrhœa they are often almost entirely composed of serum.

The *reaction* of the stools is generally more or less acid. An excess of fat increases their acidity, and much protein in the food gives them an alkaline reaction. The very acid motions which are caused by a large excess of carbohydrates in the diet are a common source of irritation of the skin of the buttocks. The watery motions of summer diarrhœa are distinctly alkaline.

The faint *odour* of the normal motions is often greatly altered in disease. When there is fat indigestion, or when acid fermentation is taking place from an excess of carbohydrates in the food, they acquire a very sour smell; while the use of raw meat juice or the presence of protein indigestion causes a varying degree of putrefactive odour.

Microscopic Examination of the Stools.—For ordinary clinical purposes a simple inspection of the motions is usually all that is necessary. Sometimes, however, it is desirable to examine them microscopically. The presence of ova and undigested starch granules can only be detected in this way,

and it is only with the microscope that blood and pus in small quantity can be recognised (p. 227). Microscopic examination of the fæces for fat is, however, very unsatisfactory. In order to determine not only the amount of fat, but also the relative proportions of neutral fat, fatty acids and soaps, chemical analysis is essential.

Some Symptoms of Digestive Derangement in Infancy.

Vomiting as a Physiological Act.—Nature has sought to make up to the baby for his lack of judgment in feeding, and his dependence on others for the choice of his food, by giving him great facility in rejecting from his stomach any meal that is unsuitable in quantity or quality. The vomiting of unwelcome food in infancy may therefore be regarded as to a large extent physiological. Washing out of the stomach is a much less distressing proceeding to infants than to older people, and it is often most useful in helping them to recover quickly from an indiscretion in diet. Vomiting in infants is never to be regarded as serious if the baby is thriving in spite of its continuance. It is important not to mistake the regular return of food due to rumination for true vomiting (p. 122).

Vomiting as a Sign of Disease.—Excessive vomiting, though one of the commonest signs of digestive derangement, is also frequently due to diseases of other systems.

(1) In *acute febrile* and *infective disease* vomiting is often one of the first symptoms noticed. This is so in pneumonia, in scarlet fever, and some of the other exanthemata and in some epidemics of influenza. It is also characteristic of *uræmia* and other *renal affections* and of *acidosis*.

(2) In *meningitis* and other *intracranial diseases* it is very common, and whenever we have obstinate and unexplained vomiting we should remember the possibility of its being cerebral.

(3) *Intestinal obstruction* from intussusception, peritonitis, or other abdominal diseases must be borne in mind as a possible cause of vomiting.

(4) *Reflex vomiting* is occasionally met with. It may be due to irritation from worms, to teething, or to ear disease.

(5) In older children, especially girls, *nervous* or *hysterical vomiting* without local disease occasionally occurs, and a similar

condition is not unknown in infants in connection with refusal to take food. It is often stopped by drop doses of liquor arsenicalis.

(6) In *cardiac dilatation* and *heart failure* vomiting is a grave symptom; it is especially to be looked for in cases of diphtheria.

(7) In most cases of *congenital hypertrophy of the pylorus*, and in *pyloric spasm*, vomiting is the most striking symptom.

(8) Continuous vomiting from birth of deeply bile-stained fluid, with bile-stained motions, may be due to narrowing of the small intestine with *dilatation of the duodenum*.

(9) In conditions, such as whooping-cough, which are accompanied by severe *spasmodic coughing*, vomiting is often caused merely by the violence of the cough.

(10) In many cases, however, vomiting is due to *improper feeding*, or to some form of *dyspepsia*; when this is the case it is usually accompanied by an abnormal state of the motions, wasting, and other indications of digestive derangement. The various conditions under which it is met with and its treatment will be considered later.

Intestinal Flatulence and Colic. — These symptoms frequently occur together. They may be present when there is neither vomiting nor diarrhoea, although they are often associated with these symptoms. *Flatulence in the bowel* may be caused in otherwise healthy infants by the excessive swallowing of wind (p. 127), but it usually arises from fermentation or other change in the intestinal contents with the formation of gas. It is apt to occur when there is too much incompletely digestible matter—such as starch, milk-curd, or fruit—in the food. Severe flatulent distension of the bowel is a common and distressing terminal symptom in abdominal tuberculosis, especially in young infants.

Colic may be due simply to the presence of intestinal flatulence, or of irritating substances arising from chemical decomposition, to the mechanical irritation of scybala in the large intestine, or rarely, to lead-poisoning. Often it is merely the result of chronic constipation, and it is a frequent symptom of some varieties of purpura (Henoch's purpura). It may also be caused reflexly—for example, by cold feet.

Extremely severe recurrent colic is caused by chronic obstruction of the lumen of the bowel. This is most frequently

due, in this country, to the cicatrisation of tuberculous ulcers in the bowel or to adhesions. When any condition of the kind is suspected the abdomen should be carefully watched for the occurrence of "ladder patterns" (p. 285), which indicate the necessity for a surgical operation to relieve the obstruction.

The presence of colic is easily recognised by noting the child's behaviour when an attack comes on. The pain makes him cry; and, during the paroxysms, he flexes the legs on the thighs and the thighs on the abdomen, bending the arms also and clenching the fists. After this has lasted for a short time some wind passes, and the pain is at once relieved, although it may soon return.

Although there is generally no difficulty in recognising the nature of intestinal colic, renal colic may sometimes be mistaken for it. As we have already seen (p. 137), the behaviour of the infant during rectal pain is quite different. Long-continued or frequently recurrent abdominal pains are sometimes caused by spinal caries, and these are not infrequently mistaken by the parents for colic.

The *treatment* of abdominal distension from intestinal flatulence consists usually in clearing out the offending contents of the bowel by a copious warm water enema, by castor oil, or by small doses of calomel ($\frac{1}{10}$ gr. every half-hour until 1 to $1\frac{1}{2}$ gr. has been given), and by strictly regulating the diet. As too much cow's milk is the commonest cause, the stopping of all milk for a time is the chief indication, and it usually produces a striking improvement. Salicylate of soda (1 to 2 gr.), an alkaline carminative mixture (Appendix E, Form. 21), or a few doses of pepsin or papain (Form. 22) are also sometimes useful. In cases of abdominal tuberculosis the symptom may be very difficult to relieve; its chief treatment consists in careful regulation of the diet.

The best immediate treatment for a severe attack of ordinary colic is a dose of castor oil and a large warm water enema or irrigation. A hot poultice or fomentation may also be applied to the abdomen, and the feet warmed. Twenty drops of brandy or a dose of carminative (tinct. carminativæ 1 minim, glycerine 5 minims, aq. ad 1 drm.) may also help to relieve the pain. If the bowels are habitually constipated this must be attended to.

In the case of healthy babies who are swallowing large

quantities of wind, atropine (in doses of 1 minim of a 1 in 1000 solution) may be given with each bottle.¹

In obstinate recurring colic in older babies small doses of codeine ($\frac{1}{32}$ to $\frac{1}{24}$ gr.) are occasionally useful as a temporary palliative while the diet is being regulated. The alleviation they afford encourages the mother to persevere. When the colic sets in immediately after taking the bottle, $\frac{1}{2}$ -drop doses of liq. arsenicalis, before each feed, are sometimes useful.

Diarrhœa.—This term is used for cases in which there is increased peristalsis and consequently abnormally frequent action of the bowels. It is sometimes applied also to cases in which the motions, though not increased in number, are very abnormal in character.

Diarrhœa, like vomiting, may be in a sense physiological, being the natural effort of a healthy body to get rid of substances which are irritating the bowel and threatening to set up disease. Like vomiting also, it may be caused by various general diseases, although it usually depends either on improper feeding or on some disease of the intestine.

(1) Diarrhœa is a common symptom in *severe toxæmias*, as in scarlet fever, measles, pneumonia, septicæmia, acid-intoxication, and uræmia.

(2) It is sometimes purely *nervous* and *reflex* in origin—no more organic than sea-sickness. It may, for example, be set up suddenly by a chill or wet feet, or it may arise from emotional causes or from teething. Lienteric diarrhœa (p. 313) occurs sometimes even in young babies. It is not uncommon for the motions to be loose and frequent in breast-fed infants during the first month or two of life. This is probably of a nervous nature, and as long as the weight is steadily increasing does not require any interference.

(3) Diarrhœa may be due to the *mechanical irritation* of undigested vegetable matters in the child's food, such as vegetable seeds and skins, coarse oatmeal or unchewed pieces of potato or apple, or to intestinal parasites.

(4) In other cases the local irritation, which determines it, is of a *chemical nature*, the commonest cause being the fermentation of carbohydrates in the bowel by acid-forming bacteria. The products of these organisms set up increased peristalsis,

¹ Sidney V. Haas, "The Hypertonic Infant," *Amer. Journ. Dis. Child.*, 1918, xv., 5, 323.

the bowel itself not being diseased. Such diarrhœa may follow the addition of too much sugar to the bottle or the giving of contaminated milk. It is characterised by acid, loose, green stools. A somewhat similar type of diarrhœa arises, much less frequently, from putrefaction of the proteins in the bowel. In it the stools are alkaline in reaction, watery, brown, and offensive.

(5) Another group is that of the *infectious diarrhœas*, which differ essentially from the last in the fact that, in them, the bowel itself is not merely irritated but diseased, owing to the action of the micro-organisms which are the cause of the illness (*e.g.*, streptococcus, dysentery bacillus, or gas-bacillus).

(6) Diarrhœa is also a common symptom of ulceration and other *organic diseases* of the intestine.

(7) The diarrhœa in ileo-colitis is specially characteristic—though the motions are frequent they are small and accompanied by much straining.

Constipation.—Constipation is a common and sometimes a very troublesome symptom in babies. It may be said to exist in any case in which the stools are harder, drier, and less frequent than they should be. This is more common in hand-fed babies when the diet is rich in salts and leads to the formation of an increased quantity of soaps. It is important, however, to remember that mere infrequency does not constitute constipation if the stools are perfectly normal; and also that some constipated babies have three or four small hard dry motions in the day.

The *causes* of constipation are many and various.

(1) *General causes.*—In some cases there seems to be a hereditary predisposition, as one or both parents are similarly affected. Anæmia is another predisposing cause, and pyrexia generally tends to diarrhœa, though it may be accompanied by constipation. Obstinate constipation is an almost constant symptom in tuberculous meningitis and some other brain diseases. Cretin babies are generally constipated, and it is possible that scanty thyroid secretion may sometimes play a part in causing this symptom in other children.

(2) *Anatomical causes.*—It is often said that the structural peculiarities of the infant's alimentary canal—the greater length of the intestine, especially of the sigmoid and of the mesentery—favour the occurrence of constipation. In a few cases the defective action of the bowels is due to some more definite

abnormality. There may, for example, be a congenital constriction of the rectum, peritoneal adhesions, or congenital hypertrophy of the colon, or even a tumour of some sort in the pelvis or abdomen.

(3) *Spasm of the sphincter* from fissure, from the irritation of scybala, or from any other cause, acts strongly in many cases in preventing the regular evacuation of the bowels. "A very tight sphincter is the cause of constipation in a small proportion of nurslings; and before beginning other treatment in such cases the sphincter should be stretched by passing a protected index finger into the rectum."¹ This simple proceeding is often very successful.

The commonest and most important causes of constipation, however, are general atony, improper feeding, and defective training.

(4) *Muscular flabbiness of the bowel and of the abdominal wall* from rickets, debility, and lack of exercise are strongly predisposing causes; and the local weakness which follows acute attacks of colitis is often followed by obstinate and prolonged constipation. A habit of constipation is always likely to be lessened by any therapeutic measures which improve the child's general vigour and tone.

(5) *Dietetic causes.*—(a) A scanty supply of food, either from the breast or bottle, always tends to the production of small dry motions; and too little fluid may have a similar effect. (b) When the food is much diluted and contains too little fat it is so completely absorbed that too little residue is left to stimulate the colon, and constipation naturally follows. (c) Too high a proportion of fat is a frequent cause of constipation, owing to the large amount of soap formed in the bowel leading to dry crumbling stools. (d) An excess of protein tends to slow the intestinal peristalsis by counteracting fermentation; and (e) a large proportion of calcium and magnesium salts may also have a constipating effect by increasing soap formation. (f) Too much starch, (g) too large quantities of boiled milk, and (h) too little fruit and vegetables, are other frequent causes of sluggish bowels in older infants.

(6) *Psychical causes.*—While giving careful attention to the general and local condition of the infant's body and to the composition of his diet, we must not forget that the movement

¹ C. G. Kerley, *Practice of Pediatrics*, Phila. and Lond., 1914, 236.

of the bowels, like other habits, is much influenced by the child's mental processes.¹ The young baby has almost everything done for him by his mother or nurse. Defæcation is one of the very few things he has to do for himself; and if he is of a neurotic constitution he will early recognise that any failure in this is a matter of concern to his elders. His perception of this fact, even at a very early age, brings with it the temptation to make a sensation by refusing to do what is expected of him, and this is a frequent contributory cause of constipation.

From the early months of life the infant should be regularly set on the chamber immediately after his morning bottle. When this is done as a routine, the normal action of the bowels generally soon follows as the result of unconscious suggestion, and its performance assumes the characters of a fixed habit. In babies, as in adults, concentration of the mind on this function is rather apt to develop doubt as to whether it can be performed, and so to inhibit its occurrence. Also, if the child realises the strong impression on his seniors that failure to perform this duty produces, the action becomes at once much more difficult to him. For this reason the question of his difficulty should never be made much of or spoken about in his presence; and his mother and nurse should aim at suggesting to him in a cheerfully optimistic way that, of course, the normal act will come off all right, and that, if it does not, no one will be much put about. Early training on these lines is very important, and in many cases it is the only thing needed to establish regular habits.

Treatment.—Our measures for the treatment of a case of constipation in a baby must depend on the causes of the condition. The use of drugs is only occasionally essential; in many cases they do nothing but harm.

(1) If the child is *lacking in vigour and tone*, or is anæmic or rickety, these conditions require special care; a laxative iron mixture (Appendix E, Form. 24) is frequently successful, and cod-liver oil may be indicated; and skilled massage, if it can be obtained, is very useful.

(2) If *local abnormalities*, such as an anal fissure, are present, they call, of course, for special treatment (p. 138).

(3) *Diet.*—The child's food must be adequate in amount, and its constituents present in approximately proper propor-

¹ H. C. Cameron, *The Nervous Child*, London, 1919, 30 and 80.

tions. It must not contain either too little or too much fat. In many babies what is needed is an increase of sugar to make the reaction of the bowel acid and prevent the excessive formation of soap in the motions. Sometimes the substitution of malt-sugar (in the form of Mellin's Food) for cane-sugar has an excellent effect, and barley or oatmeal water may sometimes be used with advantage in place of plain water.

In older infants more solid food in the diet may be what is needed, or an increase of broths and soups with a lesser quantity of milk. In other cases oat-flour or oatmeal may advantageously take the place of more constipating varieties of farinaceous food. In young babies the administration of a teaspoonful or two of orange or prune juice twice daily, or dilution of the milk with water in which vegetables have been boiled, is sometimes useful; while, in the older ones, larger amounts of these things, or apple sauce, or some form of vegetable pulp (carrot or spinach) may often give good results.

(4) *Medicines*.—Laxatives should not be used regularly for long periods in infants. Their chief value is that they can be made to assist in inducing the baby to form regular habits. In atonic infants, when the motions are fairly normal in consistence, small doses of tincture of nux vomica (1 minim) or of belladonna (3 minims) are often helpful; and milk of magnesia (1 to 2 drm.), phosphate of soda (10 to 60 gr.), phenolphthalein, cascara, grey powder, and liquid paraffin may, in different ways, exert a useful effect in the treatment of constipation by rendering the motions softer and therefore more easily passed. At the same time they increase the natural stimulation of the bowel which the baby has got into the habit of ignoring. Such purgatives as calomel and castor oil should not be given regularly for constipation.

(5) *Local mechanical treatment*.—Local stimulation of the rectum by the insertion into it of an oiled roll of paper, a piece of soap, or a gluten or glycerine suppository, is usually efficacious in producing a motion. But if a device of this kind is made use of habitually for any length of time, the infant soon gets to depend on it entirely and to ignore Nature's gentler intimations. The beneficial effect of dilating the rectum in cases in which the constipation is due to narrowing has been already mentioned.

In obstinate cases a small soap and water enema, or the

injection of a teaspoonful of glycerine, may at times be advisable; but the habitual use either of suppositories or enemata (especially those containing glycerine) is apt in time to set up irritation of the rectal mucous membrane, and sometimes causes a fissure. Occasionally, also, the injection of $\frac{1}{2}$ to 1 oz. of olive oil, gently introduced into the bowel at bedtime (through a catheter) and retained till morning, is of great benefit in the early treatment of bad cases in young infants, as well as in older children.

(6) *Psychical treatment*. — The frequent importance of suggestion in the prevention and treatment of constipation in infancy has already been sufficiently emphasised.

Constipation in older children will be considered in Chap. XII, p. 294..

CHAPTER VII

BREAST-FEEDING

Human Milk, its Composition and Character

THE colostrum, which the breasts supply in small amount during the first few days, is yellowish in colour, shows a higher percentage of protein and salts than the ordinary breast-milk, and has an alkaline reaction. It has been shown to possess for various pathogenic organisms a greater opsonic power than the milk which is secreted later, and it is believed to exert a laxative effect on the bowels. There is no doubt that it is good for the child's health.

When considering the composition of human milk it must be remembered that variations, especially in certain definite directions, occur from time to time. These may be observed during the course of the day and even during the course of a single feed. Just as in the case of cow's milk, human milk is richer in fat in the morning than in the evening and at the end of a feed than at the beginning. These are points which must be borne in mind when one decides to obtain a sample for examination. It has recently been shown by Telfer¹ that the milk of underfed and unhealthy city poor mothers may contain a diminished amount of fat and a less proportion of lime, phosphorus and iron than the milk of healthy rural mothers. The fall in fat, however, never reaches a pathological level and the supply of lime and phosphorus always remains in excess of the infant's needs, but in consequence of the deficiency of iron, which normally is barely sufficient to supply the requirements, anæmia may result.

With the above exceptions the composition of the milk is not subject to serious changes, and it may be accepted that the one day's supply is fairly constant in its composition. So true is this that it may be taken as a safe rule that if a child at the

¹ S. V. Telfer, *Biochem. Journ.*, 1924, xviii., 809; *Glasg. Med. Journ.*, 1930, cxiii., 246.

breast is not thriving it is most likely that an insufficient quantity is being supplied. It is indeed one of the most difficult of tasks to modify the milk except in so far as its quantity and fat content are concerned. These two factors tend to vary inversely.

Contrary to what might have been expected, there is little change as lactation proceeds, and hence it is not necessary, as is often suggested, that the child of the wet-nurse should be of the same age as that of the foster-child she has to suckle. It is also a common belief that mother's milk undergoes a change during the menstrual period. Of this, however, there would appear to be little evidence, as the vast majority of infants do not seem to be the least upset at this time.

It has been said that deficient exercise and over-feeding of the mother induces a high protein content of the milk, but of this there is no proof. In any case it is exceedingly doubtful if a high protein diet ever disturbs the child. The success with undiluted cow's milk in infant feeding is entirely against such an assumption. Diet would seem to have little effect on the composition of the milk, and it is not improbable that when such a regime does cause trouble it is in consequence of a diminution of the quantity.

It is generally held that the milk of cows which are stall-fed, or receive very little fresh food, is deficient in the anti-scorbutic vitamin, and by analogy one might surmise that the same could happen with human milk. That such a deficiency, however, can ever reach a dangerous level is rendered unlikely by the extreme rarity with which breast-fed children develop scurvy, and that even in countries where the disease is endemic among the adult population.¹

Although there are no definite secretory nerves, and secretion persists after all nerves to the mammæ are cut, there is no doubt that these glands are under the influence of nervous and psychic factors. Excitement and anxiety may cause the milk to disappear. If the mother is afraid that she is not going to be able to suckle, the supply of milk may fail. Under the influence of grief the same result may happen, and in South America a method of deliberately causing the milk to disappear is by inducing disgust through putting a puppy to the breast. On one occasion we have seen the composition

¹ A. F. Hess, *Scurvy Past and Present*, Phil., 1920, 37.

of the milk altered as the result of nervous factors. This was the case of a woman with syringomyelia which involved one mamma: the milk secreted by this mamma had the salt content raised to 0.37 grammes per cent. and every time the child drank from that breast he vomited, whereas he took well from the other breast and was never upset.

It is possible, of course, that certain constituents of the diet, or drugs which the mother is taking, may pass over into the milk and upset the child. This, however, is not in the true sense of the term a change in the composition of the milk. It is simply that some toxic substance, or some substance which is toxic for the child, is superadded. If, for example, the mother is taking bromides, the child at the breast may develop a rash. Opium and certain purgatives when taken by the mother may affect the child through the milk. An anaphylactic infant, too, may present various toxic manifestations if the mother is taking foods (fruits, fish, or tinned articles) to which he possesses a susceptibility.

The quantity of milk secreted is also subject to variation. It is certain that the child does not always require the same amount of milk and that the mammæ vary their response to the child's needs. Increased suction causes an increased secretion of milk. In fact, this stimulus is the only real galactagogue. Langstein and Meyer¹ have shown that a healthy wet-nurse, if required to suckle two children, or even three as has happened, responds to the increased demands by doubling or trebling her supply of milk. Even the virgin mamma can be made to react by putting a child to it. This David Livingstone observed among the native races in Africa, and he records that a father, who had been left with his infant child and no means of nourishing it, applied it to his own breast and induced the flow of milk.²

The average composition of human milk is given in the Table overleaf. It will help to impress the facts on the mind of the reader, as well as being convenient, if at this stage we compare it with cow's milk, which is so commonly employed in substitute feeding.

¹ L. Langstein and L. F. Meyer, *Säuglingsernährung und Säuglingsstoffwechsel*, Wiesbaden, 1910, 47.

² David Livingstone, *Missionary Travels and Researches in South Africa*. John Murray, London, 1877, p. 126.

Comparative Compositions of Human and Cow's Milk.

	Human Milk. Average per cent.	Limits of Variation in Normal Human Milk in percentage.	Cow's Milk per cent.
Water	88.05	89.82 to 85.50	87.4
Fat	3.50	5.00 „ 3.00	3.0 to 4.0
Protein	1.25	2.25 „ 1.00	3.5
Sugar (Lactose)	7.00	7.00 „ 6.00	4.5
Salts (total)	0.20	0.25 „ 0.18	0.75
CaO	0.045	...	0.17
P ₂ O ₅	0.048	...	0.24
Fe ₂ O ₃	0.0001	...	0.0001
MgO	0.006	...	0.02
Na ₂ O	0.0049	...	0.04
K ₂ O	0.069	...	0.18
Cl	0.029	...	0.08
SO ₃	0.0143

The constituents of the two kinds of milk differ not only in percentage composition but also to some extent in chemical and physical characters. Of these the following are the most important.

In the two kinds of milk the fat differs little in amount, but that in human milk contains more oleic acid, has a lower melting-point, and is in a state of finer division. These characters make it more easy to digest.

In both kinds of milk the protein element consists in a mixture of two substances—casein (or caseinogen), which is curdled by rennet but not by boiling, and lactalbumin, which is precipitated on boiling but not on the addition of acids or rennet. In human milk the casein is said to be to the lactalbumin, roughly, in the proportion of 1 to 2; while in cow's milk the proportion is something like 4 to 1. This difference in the relative proportion of casein and lactalbumin in the two milks is responsible for important physical and physiological differences. The relatively rich casein content of cow's milk results in the amount of curd being something like five times as bulky as that of a similar quantity of human milk. This delays gastric digestion of cow's milk and may bring about mechanical difficulties. Digestion may be imperfect and some of the curd appears in the fæces. The biochemical differences are, however, probably more important. The lactalbumin factor contains the amino-acid (tryptophan) which is essential for

growth, so that in equal quantities of milk this element is relatively more abundant in human milk.

Cow's milk contains a much larger proportion of mineral salts than human milk — the calcium and phosphorus, for example, being four times as abundant in the former as in the latter. Iron, however, is if anything a little less, and magnesium distinctly less abundant in cow's milk—facts which must always be borne in mind when dilution of the milk for infant feeding is practised.

Human milk is amphoteric in reaction—slightly alkaline to litmus and acid to phenolphthalein. Cow's milk, on the other hand, at least by the time it is available for feeding purposes, is always distinctly acid.

One point of special importance is that human milk is practically sterile as it leaves the nipple, whereas cow's milk when delivered for use always contains a large number of bacteria of different kinds. It is this difference which undoubtedly accounts for the very different incidence of enteritis in the breast-fed and bottle-fed child (pp. 215 and 218).

Another important point is that the milk of healthy women who are properly fed contains in abundance the accessory food factors which are required for the infant's growth and nutrition. It has therefore anti-scorbutic properties, so that children fed on it do not develop scurvy, and the presence of vitamin A ensures satisfactory growth.

Breast-Feeding.

During the first seven to nine months of a child's life he should draw his nourishment solely from his mother's breasts. The mother's milk is the most digestible as well as the most perfectly balanced food a child can have; and when a delicate baby is deprived of breast-milk and put on the bottle he is thereby exposed to great additional risks; this should never be done without careful consideration. If a child is strong it is less important; but if he is specially weakly (*e.g.* marasmic or syphilitic), breast-feeding often gives him his only chance of life. Not only are breast-fed babies stronger and better developed than those who are bottle-fed, but they are also much better able to resist disease. It has been found that among the poor the mortality of hand-fed infants is about six times that of babies on the breast; and that among those

who are fed artificially by strangers, nearly twice as many die as among those to whom the bottle is given by their own mother. This cannot of course be ascribed entirely to the manner of feeding, as there are many other factors which in these circumstances come into play. The great danger of the bottle-fed child among the poorer classes is enteritis from contamination of the milk with infective organisms. (See Figs. 18 and 19, pp. 17 and 18).

Inability to Nurse.—Numerous investigations carried out in continental cities have shown that the proportion of healthy women in the working classes who cannot suckle their infants, when the process is properly supervised, is exceedingly small. In Glasgow and the West of Scotland, however, a considerable number of women, not only among the leisured classes, but also among those less well-favoured, either cannot feed their infants at all, or at least cannot do so for the requisite time. Of the hospital class of mother 20 per cent. are never able to suckle at all and only 50 per cent. manage to do so for nine months. Of the better-class mothers, also, 20 per cent. never supply breast-milk; 50 per cent. practise breast-feeding for a period of three months, but only 25 per cent. for the normal nine months. There is no doubt that this state of matters is not due to disinclination but to real inability, as the mother of the present day is very anxious to do all she can for her child. Unfortunately, too, it would seem that this disability, at least among the better classes, is increasing, since a comparison of the years 1917 and 1927 shows that, while in the former year 50 per cent. could continue suckling for three months, in 1927 only 20 per cent. could do so. In 1917, 20 per cent. of the better-class mothers were able to supply enough milk till the end of the sixth month, whereas in 1927 only 8 per cent. were capable. In America conditions in this respect are probably worse. Writing in 1909, Holt¹ says that among the well-to-do classes of New York and its suburbs less than 25 per cent. of the mothers have been able to continue nursing for as long as three months. The same author² in 1922, however, states that, as a result of propaganda, matters had improved considerably, but he does not supply actual figures for comparison.

¹ L. E. Holt, *Diseases of Infancy and Childhood*, New York, 1909, 168.

² *Ibid.*, New York, 1922, 169.

It is important to remember in this connection that because a mother cannot nurse her first baby she will not necessarily fail in the case of subsequent children: it may also be stated that ability to suckle the first child does not, on the other hand, ensure that she will be able to do so on subsequent occasions.

Contra-indications to Breast-Feeding.—Now and then nursing has to be forbidden because the mother is so delicate that the strain which it causes might be too much for her. In the decision of this question we must bear in mind that breast-feeding is a physiological process, and that many weakly mothers are never so well as when they are nursing, just as many women state that they were never so well as during a pregnancy. Breast-feeding should never be allowed if the mother has pulmonary tuberculosis, because the close contact which nursing implies exposes the very susceptible infant to a serious risk of infection. Nephritis and exophthalmic goitre are also contra-indications to breast-feeding. Further, the mother may have to give up nursing owing to an abscess of the breast, cracked nipples, or some other local disease. It is, however, stated by some pædiatricians that sepsis of the mamma does not preclude breast-feeding, as suckling has a beneficial effect on the local mischief, and that the few pyogenic organisms which may be present are of no danger. It may be that the baby is unable to suck owing to debility or to some deformity of the mouth or lips, *e.g.*, hare-lip or cleft-palate. In such cases an attempt should always be made to draw the milk off for him as long as the supply lasts, and to give it with a spoon, dropper, or tube. Sometimes, as he gets stronger, he learns to take the breast quite well.

General Considerations and Practical Details regarding Nursing.—As has already been indicated, there are often difficulties in getting the process of breast-feeding set agoing; and sometimes much tact and perseverance are required. If these are exercised, many mothers, who have seemed at first to be unable to nurse, can do so perfectly well.

One common difficulty is that the milk is sometimes long in coming. There are two ways in which the secretion of milk becomes established under normal circumstances. In some cases it develops slowly, and gradually increases; in others, after a period during which only a little is secreted, there is a

sudden profuse flow. We should never, therefore, give up our endeavours until we have allowed plenty of time for this flow to take place. In some instances it occurs within the first two days, oftener on the third day, oftener still not till the fourth, and not very rarely only on the fifth day. Its appearance is encouraged by the breasts being thoroughly emptied. With perseverance the mother's own baby can usually be got to do this; but, if he fails, it is a good plan, if possible, to call in the services of an older and stronger infant whose vigorous sucking will supply the necessary stimulus to the breasts.¹ When the mother is able to leave her bed early and go about her usual work, as is much oftener allowed nowadays than it used to be, this may have a good effect in starting the full flow of milk. Nevertheless, many well-to-do mothers can suckle their children so long as they are in bed during the puerperium, but on getting up are quite unfit to do so. But it is also important to remember that about the third week there is a tendency for the milk to diminish temporarily. This must not be taken as an indication to cease breast-feeding. If suckling is persisted with, the normal adequate flow will often return.

If the mother is only able to nurse for a few weeks or months, it is generally altogether good for the child that she should do so, as it tides him over the most difficult time for his digestion; and is advisable, even if the breast-milk has to be supplemented by bottle-feeding. Far from the two kinds of milk disagreeing with one another, as some used to think, the mother's milk is found decidedly to help the digestion of the other food. Even when the nipples are too small or too sore at first, these difficulties can often be got over by care and patience and the use of a nipple-shield; and the nursing, once begun, may be quite successful.

Beginning Nursing.—The baby should be put to the breast within six to twelve hours of birth, and again every six hours during the first twenty-four, and every four hours on the second day. As the milk begins to come, the intervals may be

¹ By means of the "Perfection Electric Breast-Pump" it has been found that the mammæ can be adequately stimulated from the first day of the puerperium, so that there results an ample supply of milk and the so-called "physiological loss of weight" during the first week of life does not take place. The apparatus is supplied by Perfection Manufacturing Co., 2125 East Hennepin Avenue, Minneapolis, Minn., U.S.A.

shortened. During the first twenty-four hours the child only gets a teaspoonful or two of colostrum; and in the second day not more than 2 or 3 oz. It has generally been surmised from this that Nature does not intend the child to have any food during these days, but the delay in the appearance of milk is really due to the inadequate sucking powers of the newborn infant. As previously mentioned (p. 159), sucking is the only galactagogue. When adequate suction is applied to the mamma from the beginning the secretion of milk steadily increases and the child does not lose any weight during the first few days of life. If this is not possible by the child himself, or from another healthy and vigorous child being put to the breast or by means of the electric breast-pump (p. 164), a substitute food and ample fluid must be given in order to quench his thirst, counteract depletion, and to keep the excretion of the skin and kidneys active until lactation is properly established.

If the appearance of the milk is long delayed there is a danger of failing strength from inanition. This, of course, will react on the activity of the mamma by diminishing the suction power of the infant, which, as we have seen, is the real galactagogue. Hence some form of nourishment should be given to supplement the breast until its secretion is fully established. A 5 per cent. or 10 per cent. solution of sugar in water may be used, *e.g.*, $\frac{1}{2}$ to 1 oz. every six hours during the first day and every four hours from the second day onwards. Protein milk (see p. 180) may be employed: this diet has the advantage of supplying more nourishment, of being more akin to the natural food, and it is a good method of acclimatising the infant to cow's milk.

The colostrum, as mentioned above, acts as a laxative; but if, by the end of the first twenty-four hours, there has been no action of the bowels, it is generally wise to administer a teaspoonful of castor oil to clear out the meconium, because its retention occasionally gives rise to nervous symptoms and may be responsible for prolonging the manifestations of icterus neonatorum, so that the possibility of obliteration of the bile-ducts may be raised.

It is important that the child should be put to the breast regularly and at the same hours every day; but no hard and fast rule as to the length of the intervals can be laid down.

They may have to be varied according to the child's size, vigour, and appetite. It may, however, be said in a general way that for the first week the baby should be put to the breast every three hours during the day, with one long interval of six hours during the night, *i.e.*, seven times during the twenty-four hours. During the second week, if the child is drinking well, the intervals should be lengthened to four hours, with one longer interval of eight hours during the night, *i.e.*, five times during the twenty-four hours. Convenient hours of feeding are 6 A.M., 10 A.M., 2 P.M., 6 P.M., and 10 P.M. The great advantage of such a method of feeding is that the child is more likely to empty the breast thoroughly. This not only ensures a feed of average composition but at the same time a better stimulation of the gland. By the longer intervals the stomach will be empty before another feed is due, and thus a less tendency to vomit from perversion of digestion. Another important advantage of the infrequent feeds is that they give the mother the opportunity of uninterrupted sleep during the night and facilities for sharing in social life during the day.

In nursing, the breasts should be given to the child alternately. He generally takes about twenty minutes to a meal, although the bulk of the feed is obtained during the first five minutes. This is particularly true of the vigorous child, who may get all he needs in fifteen, ten, or even five minutes. If he takes more than half an hour, or falls asleep while at the breast, there is something wrong: either he is weakly or there is too little milk for him. If one of the breasts is smaller and contains less milk than the other it is especially important that it should be used as much as the other, since it is only in this way that it can be stimulated to increased function.

Care of the Nipples.—Towards the end of pregnancy the mother's nipples should be gently manipulated and drawn out so as to prepare them for nursing. When lactation begins they must be carefully washed and dried after each nursing, and methylated spirit may be applied to harden them. If cracks appear they should be treated with a simple ointment (Appendix E, Form. 58), and a nipple-shield used during nursing. If the secretion of milk is threatening to fail it may sometimes be re-established by emptying the breast regularly with a breast-pump. The electric pump is said to be specially valuable for this purpose.

Defects in Breast-Feeding.—The commonest fault in breast-feeding is an insufficient supply. The child may be quite contented but he does not put on weight. In another case he is restless, always crying, fights with the nipple during the feed, and loses weight.

In order to decide definitely how much milk the child is getting he should be weighed immediately before and after a feed—the increase in weight represents the amount of milk consumed. When the breast-milk is not sufficient it should, as already said, be supplemented by artificial feeding—*allaitement mixte*. The younger the child the more important is it that this procedure should be adopted rather than that he should be weaned. The breast- and bottle-feeds may be given alternately, but the preferable method is to put the child to the breast, or even to both breasts, at every feed and to make up the deficiency by a bottle-feed as calculated from the child's requirements (p. 176) and the amount of breast-milk taken, as determined by weighing before and after the breast-feed. The child requires, for example, $3\frac{1}{2}$ oz. of milk every four hours, and from weighings before and after the feed it is found that there is only an increase in weight of 2 oz.: a supplemental feed of $1\frac{1}{2}$ oz. of cow's milk should therefore be given after each breast-feed. Since the amount taken at a feed varies somewhat, it is needless to point out that the weighings should be carried out after several feeds and an average figure obtained.

As the stimulus of sucking is the only real galactagogue, it is advisable at the same time to diminish the intervals between the feeds, *e.g.*, to three hours. After some time of this regime it may be found that, the supply being sufficiently increased, one is able not only to discard supplemental feeds but also to revert to the four-hourly intervals.

If the secretion of milk is too profuse, or the child greedy, he will bring up some of it shortly after each nursing (*possetting*) and he may have too many stools. In this case the duration of the feed may be shortened, but as long as the weight is steadily increasing there is no cause for anxiety. It should always be remembered that it is not uncommon for a highly nervous child to have frequent loose stools during the first three or four weeks of life: this condition usually rectifies itself, and so long as the child is otherwise well there is no call for any alteration of regime.

In a child not thriving at the breast the possibility of something being wrong with the milk is the last thing to be thought of. If, however, it is decided that the milk should be examined, then a sample about the middle of the feed must be selected, or better, a sample of the total secreted during twenty-four hours. But, as mentioned above (p. 157), the composition of milk is very constant and is difficult of modification. Sometimes a milk which seems quite unsuitable on examination is found in practice to agree perfectly well. It is doubtful if the reputed causes (deficient exercise, menstruation, and over-feeding) of change in the composition of milk have any foundation in fact: some change in quantity is more likely to be the underlying factor. The success with which the poorer classes practise breast-feeding and the want of success of the better-class mother would seem to dispose of dietetic and hygienic factors playing any significant rôle. In the under-nourished city mother the fat content may be slightly deficient, but not to pathological levels; as previously mentioned, the deficiency of iron, which even in normal milk is so close to the actual requirements, is a much more serious matter.

If there is sufficient milk and the child is not thriving then the fault lies with the child. There may be tuberculosis, otitis media, pyogenic infection of the urinary tract, or pyloric stenosis, and a careful physical examination is called for to discover if one of these conditions is present.

An early tuberculous infection may be responsible for the child failing to gain in weight. Physical examination may fail to detect any disease, and it is in these circumstances that the tuberculin reaction is of such importance (p. 946).

Diarrhœa and vomiting in the breast-fed infant are often due to pyelonephritis or otitis media. Vomiting, alone, may be caused by some congenital stenosis of the œsophagus, hypertrophic pyloric stenosis, or pylorospasm.

Constipation, although less common in the breast-fed than in the bottle-fed child, is, nevertheless, not very infrequent. The safest method of relieving it is to administer a laxative carbohydrate such as malt extract or milk of magnesia. The dose required varies, and must be found out by the mother herself. A safe rule is to commence with half a dram thrice daily and increase or diminish as necessary. Simple remedies such as these are preferable to the use of stronger purgatives

or of suppositories and enemas. It should be taken as a golden rule never to give castor oil for constipation, as owing to its later constipating effect this drug invariably makes matters worse.

Weaning.—A child should not be taken off the breast before the usual time except reluctantly and for very sufficient reasons. The reappearance of menstruation does not usually make weaning necessary or desirable, for in most cases the baby is not upset by its onset; and, even if he ceases to gain weight for the time and has colic and undigested stools, this need not do him much harm, and he can be hand-fed for the few days that it lasts.

If pregnancy occurs it is wise gradually to wean the infant, although many women quite successfully suckle the infant during the first few months. During an acute illness on the part of the mother, *e.g.*, influenza or pneumonia, the weaning should only be temporary. During the acute stage of the disease the breasts can be kept functioning by emptying them with the breast-pump and breast-feeding resumed on convalescence. The age of the infant is, of course, an important element in deciding the conduct of the case.

Under ordinary conditions the baby should be weaned when he is between nine and ten months old. By this time he needs other food than milk. The small iron content of milk makes prolonged milk feeding lead to anæmia. If possible, the child should not be weaned during the heat of the summer unless the greatest care can be taken in the details of hand-feeding, on account of the susceptibility of the infant to enteritis.

Weaning should always if possible take place slowly, for the sake of the mother as well as for that of the child. It is easier for the baby if he has been long accustomed to take one bottle a day while on the breast; and this practice is good for the mother also, as it allows her more freedom and encourages her to persevere with the nursing.

When babies are weaned early, they should be given the artificial food from a bottle, but older infants should have it from a cup or spoon. To begin with, the food should be rather weaker than would be given to a bottle-fed baby of the same age, so as to lessen the risk of indigestion from the change.

Wet-Nursing.—When the mother is unable to suckle her child she must either get a wet-nurse or begin hand-feeding.

For various practical reasons wet-nurses are rarely employed nowadays in this country for healthy children. They are not only difficult to find and expensive to keep, but the circumstances which induce them to offer their services are often such as to render them undesirable inmates of a house.

While wet-nurses, therefore, are rarely used now for healthy babies, it must always be remembered that no form of artificial feeding, however skilfully planned and carefully carried out, is nearly so good for weakly children as suitable wet-nursing. Many infants who are dying of atrophy, or from the exhaustion caused by severe diarrhœa or other disease, may be saved by it and by nothing else. Where the infant is too weak to be able to draw the milk from the wet-nurse's breast it should be taken off for him by a breast-pump and given through a bottle or with a syringe. In Boston, U.S.A., an association has been formed for the collection of breast-milk from women who have more than they require and which is distributed to necessitous cases by a medical board.¹

It is often a good plan to get the wet-nurse to bring her own baby with her, because he may be a great help in keeping up the secretion of milk by thoroughly emptying the breasts. If there is proper supervision there should be no danger of his being cared for at the expense of the foster-child. It is not necessary, as is often stated, that the foster-mother's child be of the same age as the child to be suckled.

In the choice of a wet-nurse it is essential that she and her baby be thoroughly examined and that a Wassermann test be carried out.

¹ F. B. Talbot, *Journ. Amer. Med. Assoc.*, 1911, lvi., 1715.

CHAPTER VIII

ARTIFICIAL FEEDING

THERE is probably no more important subject in the whole range of therapeutics than the artificial feeding of infants, for there is no other by which, if properly carried out, more lives can be saved, and certainly there are few sorts of treatment the mismanagement of which has such disastrous results. Careful feeding during the early weeks and months of life prevents a large proportion of the diseases from which babies die, for it not only supplies them with the nourishment they need to grow and develop normally, but also gives them a store of strength by which they are able to resist the attacks of pathogenic organisms and other morbid influences, both during infancy and in later childhood. If no mistakes were made in feeding babies a large part of our infant mortality would come to an end.

The systems of artificial feeding of the infant are almost legion. They are for the most part based on the idea that the quantitative distribution of the various proximate principles is the important factor, and entirely ignore the fact that qualitatively the milk of each species of mammal is fundamentally different. We have already referred to this matter when discussing the digestion and utilisation of protein (p. 130). It was there pointed out that protein as such was only of value in virtue of the amino-acids of which it is composed, and that in the protein molecule these were not only quantitatively but also qualitatively very different. It is the tissues of the individual species which alone can by katabolism and anabolism change the structure of the protein molecule. We have also seen that the salts are present in varying proportions in the different milks and to bring these even into line would be a herculean task. The lesson to be drawn from these facts is that Nature has supplied the young of each species of mammal with a food specially suited for its particular needs, of which

rate of growth may be the most important. They also impress on us the advisability of practising breast-feeding at all costs, and they dispose of the conception that any variety of milk can by human effort be so transformed as to be in any way like human milk.

Choice of a Substitute for Breast-Milk.

All mothers, however, cannot feed their children, and, as in this country the provision of a wet-nurse is practically impossible, some substitute must be found. It is only natural that we should select some similar food supplied by Nature for the sustenance of the new-born. Hence milk of some mammal is usually employed. Cow's milk, ass's milk, and goat's milk have all been tried and are quite serviceable, but cow's milk, being the one that is alone procurable in sufficient amount, is the only variety that need be seriously considered for general use. There is, however, no special reason why one variety should be generally preferred. They all differ, as we have just remarked, from one another in composition. Nevertheless, there are occasions when one or other variety is essential. Very rarely the infant possesses an *idiosyncrasy* to cow's milk, just as individuals occasionally possess an idiosyncrasy to certain articles of food, or the seeds of certain grasses, so that they develop, on ingesting the one or coming in contact with the other, skin rashes, asthma, or inflammation of the nose and eyes (hay fever). The infant, on the ingestion of a small amount of cow's milk, may develop severe vomiting and diarrhoea and suffer from such profound collapse that he looks as if dying. In these circumstances it is necessary to procure either goat's or ass's milk until the child has been gradually acclimatised to cow's milk.

One marked difference between natural and artificial feeding is that by the former method the child is getting direct from the mamma a living substance, whereas by the latter he is given a food which is not a living substance. Apart from the activity of the vitamins this is believed by some to be of importance. But the real danger of cow's milk is that it may convey infection. There is little doubt that, so far as the poorer classes are concerned, this is the cause of much trouble. Breast-milk is almost invariably a sterile food, whereas a sterile cow's milk is unknown. When one recollects that milk is a good medium for the growth

of organisms and that the milk consumed on one day, especially in a large city, is often that which has been obtained from the cow the day previously, and when one visualises the conditions under which it is kept in the homes of the poor, there is little wonder that artificial feeding is such a dangerous proceeding. The gastro-intestinal tract of the infant is exceedingly delicate and milk which the adult can take with impunity may, and frequently does, cause a fatal enteritis in the infant.

The milk may be contaminated at the source from disease of the cow, or want of cleanliness of the udders and methods of milking. Tuberculosis of the udder leads to the presence of the *tubercle bacillus*, and septic infection to the presence of *streptococci*, and contamination by the excreta to the *Bacillus coli communis*. During its transit from the source to the consumer there are further opportunities of its becoming contaminated, and if the worker be suffering from ambulatory enteric fever, diphtheria, or scarlet fever, it may be infected with the organisms causing these diseases. There is no doubt that epidemics of these various diseases have been caused by milk.

In view of all these dangers it is not surprising that public health authorities and all those interested in public health insist on the necessity of a pure milk supply. Undoubtedly matters are better than they were, but unless we can guarantee the purity of the milk, *i.e.*, that it has been obtained from tuberculosis-free cows, has been obtained by artificial milkers and has been cooled at once and bottled at the farm, it is unwise to use for infant feeding milk which is not sterilised by boiling or pasteurisation. There is good reason to believe that, in addition to making the milk safe for the infant's consumption, boiling or pasteurisation renders it more digestible. Some authorities, however, hold that sterilisation of the milk exerts a deleterious effect, but the only undoubted one is the possible destruction of the anti-scorbutic factor. This is not a serious matter, as the evil consequence from such a result can be easily avoided by the administration of a small amount of fresh orange or lemon juice.

Modification of Cow's Milk.

Other than by making the milk safe for consumption by pasteurisation or boiling, no modification of the milk is necessary. As already mentioned, owing to the fundamental differences in

the chemical composition of the protein of cow's and human milks, it is out of the question to think of making the one in any way resemble the other. The relatively larger amount of protein in cow's milk has, however, always been the stumbling-block. But when we appreciate that bulk for bulk cow's milk protein contains only one-half the amount of tryptophan which is present in human milk protein, and when we realise that this amino-acid is essential for normal growth, the increase in the protein content of cow's milk is a distinct advantage. Dilution of the milk, so frequently recommended, would reduce the quota of this very essential amino-acid. This procedure at the same time would reduce the proportion of the equally necessary salts, some of which, *e.g.*, iron, in the amounts present in undiluted milk, are barely sufficient to cover the child's needs. The proportions of sugar and cream are also reduced. By the addition of sugar, cream, and some preparation of iron these defects of the diluted mixture could be remedied, but there would still be left the serious protein deficiency. In any case, it has still to be proven that an increased protein-content of the diet, at least within limits, exerts any deleterious effect. It is said by the adherents of this doctrine that it is not at the time that the evidence of damage declares itself, but later on in the form of hepatic and renal cirrhosis. In our experience a high protein diet, *e.g.*, the protein milk of Finkelstein, is specially well borne in any catarrhal or damaged state of the bowel, probably on account of its exerting an anti-fermentative action.

It is now generally, although not universally, admitted that the infant, and even the new-born infant, can digest and assimilate undiluted cow's milk. Budin, the French obstetrician and pioneer in infant welfare, was the first in modern times to recommend undiluted cow's milk. He showed that infants did better with undiluted than with diluted milk.

At a later date, Sir Almroth Wright suggested that the addition of citrate of soda (1 to 2 gr. per oz.) to cow's milk, by precipitating some of the calcium, would render the curd less tenacious and thus more like the curd of human milk and more easily digested. Others again recommended the addition of an alkali to neutralise the acidity of the milk, and the most popular preparation was lime water, and in some cases this or soda bicarbonate would seem to be of benefit.

Still more recently, Marriott has recommended acidifying

the milk, and for this purpose a weak acid (lactic acid) is selected. For the preparation of this milk Marriott gives the following directions: "One quart of cold pasteurised or boiled milk is placed in a bowl and $1\frac{1}{2}$ drams of lactic acid (75 to 85 per cent.) is added, drop by drop, while the milk is stirred. The first half of the acid may be added very rapidly, but the remainder must be added slowly. Agitating for a few minutes with a Dover egg-beater is advantageous, but not essential. It is important that the milk be thoroughly cold before the acid is added." The added acid is supposed to spare the acid of the gastric juice and thus to exert a so-called "buffer" or saving action. In our experience no better results are obtained with lactic acid milk than with ordinary milk; in fact, the increased acidity would appear not infrequently to induce vomiting.

Quantity of Milk.

The all-important point in feeding with undiluted milk, or with any milk mixture, is that a sufficiency be given, and one feels that much of the advantage of undiluted milk is that with it underfeeding is less likely. This question of estimating the amount of nourishment would appear to be the greatest stumbling-block in the whole subject of infant feeding. We think it may safely be said that non-attention to this factor more than to any other is the cause of much of the disease during infancy. By attention to this matter it is possible to hinder many well babies from becoming ill. The wasting in consequence of starvation is itself sufficiently grave, but by rendering the child more susceptible to infection (otitis media, bronchopneumonia, and pyelonephritis) a hopeless and fatal state of matters is not infrequently produced.

Food sufficient for the natural functions, to repair wear and tear, to allow of growth, and incidentally to provide for waste, must be supplied, and unless this is done only one result can ensue, and a result which is commensurate with the shortage. If there is supplied only enough for the vital functions and to repair wear and tear, there certainly will be no growth, but there may be no loss of weight, and even with less than is required for these purposes the metabolic processes may work at a slower rate, so that there is still no loss in weight. If, however, there be not supplied sufficient for these various purposes, then the infant will live on his own tissues and wasting is the inevitable result.

Estimation of Amount of Food.—The earliest estimates of how much food the child should get were made from a study of the amount taken by a healthy breast-fed infant. The infant was accurately weighed before and after each feed, and the increase in grammes represented the number of cubic centimetres of milk consumed. This method has been employed by several workers, some of them, like Reyher,¹ making observations on their own children. Camerer and Feer and Meyer have also supplied us with figures, and from the various studies the average daily quantity consumed by the normal infant may be said to be the following:—

1st week	0-16 oz.
2nd „	17 „
4th „	20 „
8th „	27 „
14th „	28 „
20th „	30 „
20th „ onwards	35 „

By converting these quantities of milk into their caloric values (the calorie being the unit of heat or energy), Rubner and Heubner laid down the rule that the average nutritional requirements of the normal infant were—

During the 1st 3 months 100 calories per kilo. body weight

„	2nd 3	„	90	„	„	„
„	3rd 3	„	80	„	„	„
„	4th 3	„	70	„	„	„

If one desires to work with the English instead of the metric system, then the child should receive

During the 1st 3 months 45 calories per lb. body weight

„	2nd 3	„	40	„	„	„
„	3rd 3	„	35	„	„	„
„	4th 3	„	30-32	„	„	„

Fig. 65 shows the food and caloric intake of a healthy child from six months to one year of age and supports the teaching of Rubner and Heubner.

Within recent years an attempt has been made to give

¹ L. Langstein and L. F. Meyer, *Säuglingsernährung und Säuglingsstoffwechsel*, Wiesbaden, 1910.

greater precision to this matter by estimating the heat production in calories of an individual infant during a definite period of time, or from observing the amount of oxygen consumed in the process of combustion also during a certain definite period of time, and the results obtained go in the main to support the dicta of Rubner and Heubner.

Child should be fed according to Age.—Rosenstern¹ first noticed that wasted infants often did not improve till they received nourishment proportionate to their age and thus might be taking 200 calories per kilo body weight instead of the

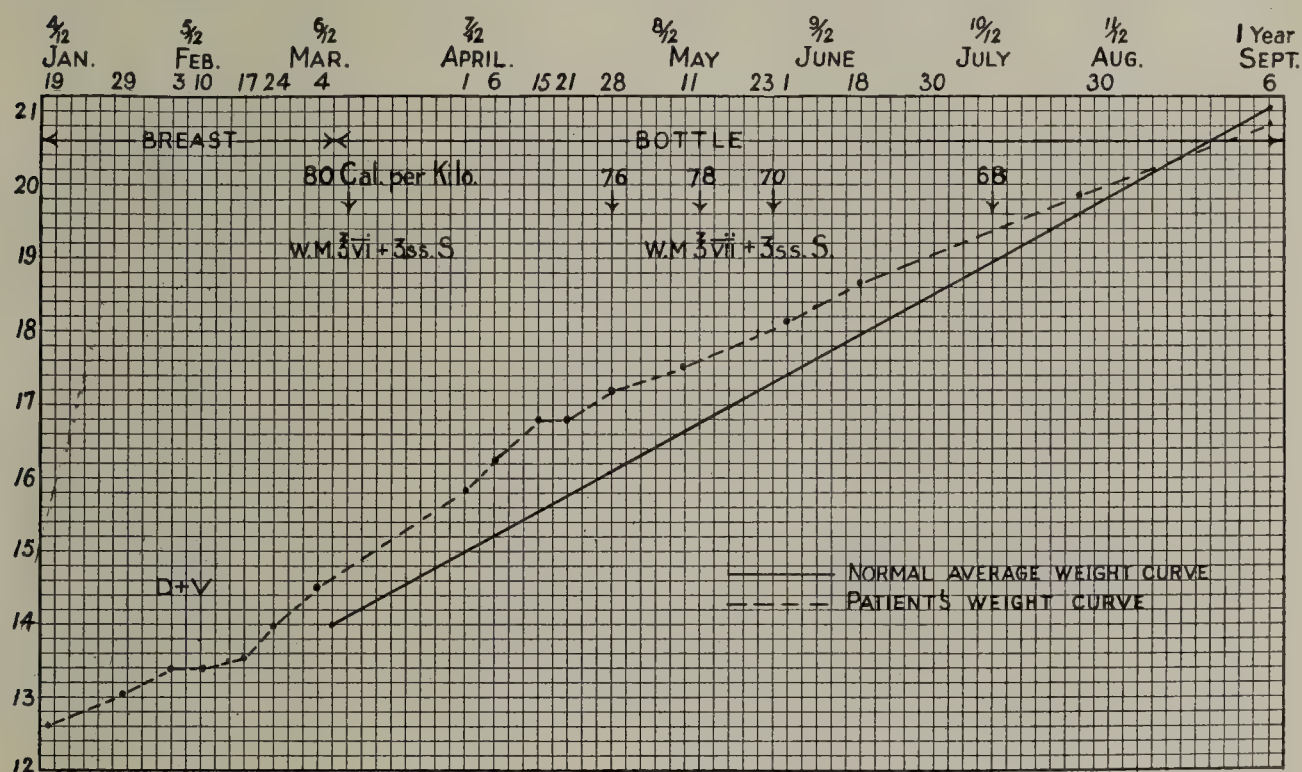


FIG. 65.—Showing caloric intake per kilo body weight from six months to one year in healthy infant.

D + V = Diarrhoea and vomiting.

normal 100. Fleming,² by estimating the basal metabolism, *i.e.*, the energy production at complete rest, in infants of varying nutritional states, found that until a child had lost 35 per cent. of the weight normal for its age (*i.e.*, its expected weight) its nutritional needs were not diminished. Hence a child, unless extremely emaciated, should be fed not according to its actual weight, but according to what it ought to weigh, or in other words, according to age. If the child is much emaciated, *i.e.*, has lost more than 40 per cent. of its weight, we can commence with less than the optimum, but if there is no increase in weight

¹ I. Rosenstern, *Deut. Med. No.*, 1909. No. 43

² G. B. Fleming, *Quart. Journ. Med.*, 1921, xiv., 171.

the quantity must be increased, and of course as weight is gained the amount in any case must be augmented.

In order to impress the above facts on the reader, we will give two examples of the estimation of the amount of food required (*a*) by the normal infant and (*b*) by a child only 50 per cent. of its normal weight. The various figures which are necessary for the calculation are:—

1. The birth weight = 7 lb.
2. The rate of increase in weight = $1\frac{1}{3}$ lb. per month.
3. The caloric requirements of the normal child, viz.:—
45, 40, 35, and 30 to 32 per lb. per day during the 1st, 2nd, 3rd, and 4th trimesters.
4. Caloric requirements of under-weight child, viz.:—
1 calorie per kilo, or .5 calorie per lb. less than the normal for every 1 per cent. less than 65 per cent. of expected weight.
5. Caloric value of diet to be fed (see table on p. 180), which for ordinary cow's milk is 17 per oz. and for sugar 16 per dram; it may also be remembered that dried milks, when made up according to directions in the proportion of 1 part of dried milk to 7 parts of water, have the same relative nutritive value as ordinary milk.

Example 1.—To find the food requirements of a normal child of 4 months.

Weight at birth = 7 lb.

Weight at 4 months = $7 + 4 \times 1\frac{1}{3} = 7 + 5\frac{1}{3} = 12\frac{1}{3}$ lb.

Calories per day required at 4 months = 40 per lb.

∴ total calories required per day = $40 \times 12\frac{1}{3} = 493$.

1 oz. milk = 17 calories.

∴ Total quantity of milk required per day = $\frac{493}{17} = 29$ oz.

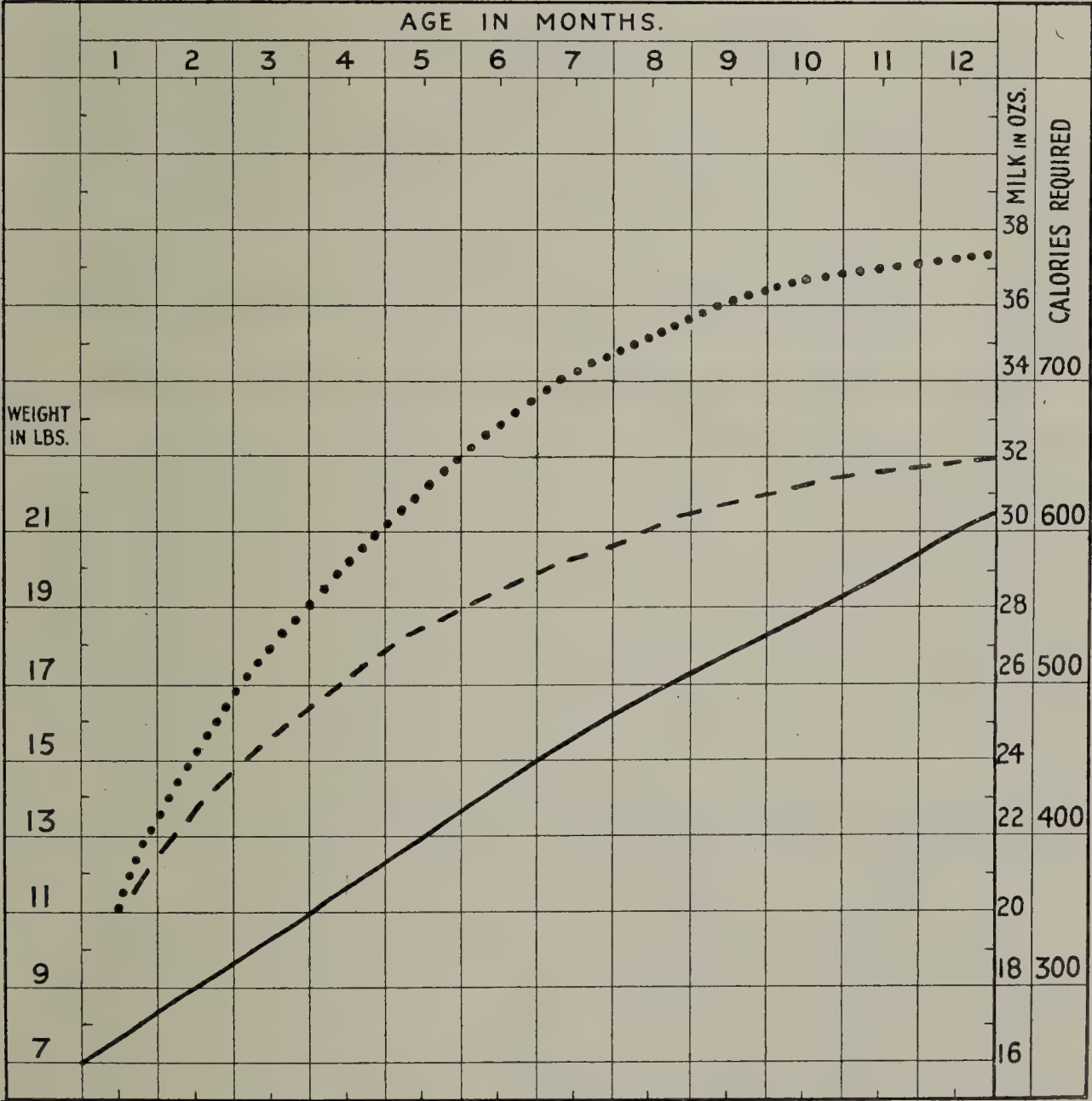
If child be given 5 feeds per day, each feed consists of $\frac{29}{5} = 5\frac{4}{5}$ oz.

If it is decided that sugar should be added to the diet in the quantity of, say, 1 dram to each feed, then as 1 dram of sugar and 1 oz. of milk have practically the same caloric value, each feed would consist of $4\frac{4}{5}$ oz. of milk and 1 dram of sugar.

Example 2.—To find the food requirements for a child of 4 months, weighing 6 lb.

- Normal weight for age = $12\frac{1}{3}$ lb.
- ∴ Child has $\frac{6 \times 100}{12.3} = 48.7$ per cent. of expected weight.
- ∴ Child should receive $65 - 49 = 16$ calories per kilo or 8 calories per lb. less than normal for age, which is 40 per lb., or $40 - 8 = 32$ per lb., calculated, of course, on expected weight.
- ∴ Daily caloric requirements = 12.3 (normal average weight) $\times 32 = 395$ calories.
- ∴ Daily amount of milk required = $\frac{395}{17} = 23.2$ oz.
- ∴ Each feed, if 5 per day, should consist of = $\frac{23.2}{5} = 4\frac{2}{3}$ oz.

or, if sugar be added, 4 oz. of milk and 1 dram of sugar.



— WEIGHT IN LBS. — — CALORIES REQUIRED PER DAY. DAILY QUANTITY OF MILK IN OZS.

FIG. 66.—The daily caloric requirements, the daily amount of undiluted milk required, and the average weight at different ages throughout infancy.

For ready reference and the elimination of the foregoing calculations the chart (Fig. 66) on preceding page, which one of us (L. F.) published in *The Practitioner*, may be found useful.¹

Caloric Values of Various Foods.

Breast-Milk	20	per oz. or 70	per 100 c.c.
Cow's Milk (undiluted)	17	„ 60	„
Milk and Water (equal parts)	8.5	„ 30	„
2 per cent. Fat Milk	13	„ 45	„
1 per cent. Fat Milk	11	„ 38	„
Peptonised Milk	13.5	„ 50	„
Lactic Acid Milk	17	„ 60	„
Albumin Milk	10	„ 35	„
Sugar	16	per dr. or 4	per gramme.

Although we believe that in the great majority of instances undiluted cow's milk is well borne by the infant, there are occasions when some modification of the milk other than by sterilisation or pasteurisation is to be recommended. This is especially the case in the new-born and in the presence of disease. The most serviceable of these modifications and their chief indications are as described below.

Protein Milk (Eiweiss Milch).—The fundamental principle underlying the preparation of this modification of milk is the reduction of the salt content so that it is brought into line with that of breast-milk. In the specially high salt content of cow's milk Finkelstein and Meyer see the chief disturbing factor. According to them the nutritive elements are not suspended in a medium which permits of the intestinal mucosa working at an optimum. At the same time the proportions of fat and of sugar are also diminished, while that of the protein remains high. In this way the anti-fermentative effect is retained and the irritating and fermentative effects of fat and carbohydrate are lessened.

Protein milk as recommended by Finkelstein and Meyer is made as follows: The curd of 1 litre of milk is thoroughly broken up and mixed with $\frac{1}{2}$ litre of butter-milk and $\frac{1}{2}$ litre of water. This gives a milk mixture of the following composition:—

¹ L. Findlay, *The Practitioner*, 1930, cxxv., 5.

Composition of Protein Milk (Finkelstein and Meyer).

Protein	.	.	3.0 per cent.
Fat	.	.	2.5 „
Sugar	.	.	1.5 „
Salts	.	.	0.5 per cent. (chiefly calcium salts).

Unfortunately, butter-milk procurable in the ordinary way in this country is not suitable for infant feeding and hence if desired must be specially prepared. This can be done by inoculating with a culture of the lactic acid bacillus, or with a small quantity of previously prepared butter-milk, milk from which the fat has been removed by skimming after it has stood for some time (4 hours at least) or by means of the separator. As both these methods require more facilities than are available in the home, it is seldom possible to procure this variety of *Eiweiss milch* or protein milk in the fresh state. It can, however, be procured in the dried state, and although we have no personal experience with this preparation, good results are reported. We have found, however, that the principles involved in the preparation of this milk mixture can be observed and equally good results obtained by the following simple method: One pint of milk is curdled with one teaspoonful of rennet and allowed to strain through a muslin cloth for 20 minutes in order to separate the curd from the whey. The curd is then broken up by rubbing through a fine hair-sieve with half a pint of cold water. In order to get as fine a division of the curd as possible this procedure may be repeated once or twice. To the suspension of disintegrated curd one-third of the total amount of whey collected is added and the whole mixture made up to the volume of one pint by the addition of cold water. Care must be taken not to exert any pressure on the curd while straining, otherwise some of the curd will escape through the muslin and there will result an excessive loss of fat and protein. When prepared as above the mixture has the following average composition:—

Protein	.	2.75 grammes per cent.	Sugar	.	1.9 gramme per cent.
Fat	.	2.28 „ „	Salts	.	0.46 „ „

This mixture should be given practically cold, or simply with the chill removed, as on warming it is prone to curdle and thus its administration by the bottle is impossible. Infants, however, do not seem to object to the cold mixture.

Protein milk is an exceedingly useful diet in any form of diarrhoea or enteritis. Its relatively high protein content and low proportion of sugar and salts exert a constipating effect. This modification of milk is also of great service in the treatment of athrepsia when the infant's tolerance to fat is lowered, and it will be found the variety of feed best borne by the newborn. In the case of the latter, when breast-milk is not available, it is better borne than any other milk mixture and is an excellent method of introducing the infant to cow's milk, with which it may be gradually replaced. So long as the motions are undigested it should be prescribed without any additional sugar, but when the fæces become normal, carbohydrate in some form (sugar—sucrose alone or a mixture of sucrose, maltose, and lactose) should be added.

Peptonised Milk.—It has always been appreciated that the curd of cow's milk is coarser and tougher than that of breast-milk, and in consequence is digested more slowly. One can also understand how the large curds may interfere with the passage of the gastric contents through the pyloric orifice. The addition of citrate of soda and lime-water was made with the intention of making the curd finer, and it is not improbable that barley-water and other farinaceous stuffs (roasted flour) exert their beneficial effect by acting as a mucilage and hindering the formation of a tough curd. Predigestion of the milk is another method of reaching the same end, and for this purpose Fairchild's peptonising powders are as a rule employed. The usual method is to mix a certain quantity (24 grains)¹ of the powder with 5 oz. of water, add this to one pint of milk, and allow the mixture to remain at blood-heat for varying lengths of time according to the degree of peptonisation desired—20 to 30 minutes is the period usually commenced with. When the degree of peptonisation desired has been obtained, the mixture is raised to boiling-point to destroy the ferment and so stop digestion.

At one time peptonised milk was a favourite remedy for all varieties of nutritional disease, but it has somewhat gone out of fashion. In our experience it has only a useful purpose in pyloric stenosis and pylorospasm. In many examples of pyloric stenosis the vomiting is to a great extent controlled by this diet, due no doubt to the fact that the removal of all curd

¹ Messrs Fairchild supply this quantity in small glass tubes.

facilitates the passage of the stomach contents through the narrow pylorus.

Lactic-Acid Milk.—Reference has already been made to lactic acid milk and the method of its preparation (p. 174). According to Marriott this modification of milk is so well borne, not only by the healthy but also by the sick infant, that it can be used as one would breast-milk.

Since both condensed and dried milks are frequently employed in the feeding of the healthy and sick infant, and in certain circumstances may have definite advantages, these preparations will next be considered.

Condensed Milk.

Condensed milk is prepared by sterilising fresh cow's milk by heat (212° F.) and evaporating it *in vacuo*. In some instances it is simply condensed (unsweetened condensed milk); in others (sweetened condensed milk), about 7 oz. of cane-sugar are added to each pint of milk. When we compare the analysis of specimens of condensed milk given by various authorities, we get the impression that the composition of the different brands varies a great deal.

Sweetened Condensed Milk.—Even when one of the better brands of sweetened condensed milk—such as Nestlé's—is used, in the proportion of a heaped teaspoonful to 3 oz. of water, the mixture only contains about 1 per cent. of protein, 1 per cent. of fat, and from 6 to 7 per cent. of sugar (mostly cane-sugar). It is therefore not sufficiently nourishing for prolonged use, and the amount of cane-sugar in it is apt to produce looseness of the bowels. When given in a much weaker solution, as it very generally is among the poor, it is extremely deficient in nutritive properties. When regularly used it is a fruitful source of infantile scurvy.

Unsweetened Condensed Milk.—Those brands of condensed milk to which no cane-sugar has been added are free from some of the disadvantages of the sweetened varieties; but they are expensive and ordinarily have no advantages over properly prepared cow's milk. They are usually not more than twice the strength of fresh milk; and once the tin is opened they do not keep longer than it does, so that they cannot safely be used after more than from twenty-four to thirty-six hours, even in

cool weather. Some specimens are sterile, but others are not so. Sugar should be added to the mixture in the same way as to ordinary cow's milk.

Uses of Condensed Milk.—Condensed milk is sometimes useful during journeys, or when the usual milk supply is temporarily interfered with; but, for the ordinary purpose of feeding infants, it is not to be regarded as at all satisfactory. The small proportions of protein and fat in it make it useful also as a temporary food in some forms of indigestion with vomiting, but the sweetened varieties are apt to cause fermentation and diarrhœa owing to their large proportion of cane-sugar. In any case it should only be used for a short time.

It is not uncommon to find that infants who have been suffering from vomiting and loss of weight cease vomiting and gain weight at once on being put on it. In these cases the cessation of the vomiting can be explained by the low percentage of protein and fat in the condensed milk, but the gain in weight is often deceptive and depends on a retention of water in the body caused by the high percentage of cane-sugar (p. 134). Children fed on condensed milk are often fat, but they are also flabby and have a low resistance to disease. If they have diarrhœa they are apt to lose weight and strength rapidly.

Dried Milk.

Dried milk consists of cow's milk which has been reduced to a fine powder by evaporation either at a moderate or high temperature. It has recently come largely into use, and is sometimes valuable in infant feeding when preparations of fresh milk cannot be got or have failed to agree, and when neither human nor ass's milk can be obtained. The varieties of dried milk most used in this country are "Glaxo" and "Cow and Gate Milk." They are apparently sterile and keep indefinitely in the tins. When diluted, in the proportion of 1 oz. of water to 1 dram by weight¹ of milk-powder, the former is said to have an approximate composition of protein 2.7, fat 3.25, and milk sugar 5.25 per cent. (Still); but some specimens have

¹ As the household tea- and dessert-spoons vary very much in size, these should seldom be used in the making of milk mixtures. With most dried milks a special measure for the correct amount of powder to be used is supplied. For the same reason the water should not be measured by spoons but by proper measures.

a higher proportion of fat. The forms of dried milk which are prepared by exposure to a high temperature for a short time are much less likely to cause scurvy than those prepared by a slower process (p. 241). In any case, it is always advisable to give orange-juice along with them. They often agree well with healthy children; and Porchat in Lyons and Eric Pritchard in London have reported good results from their use in dispensary practice. In many infants, however, they set up fat indigestion with hard white soapy stools. This is due, probably, not only to their too high proportion of fat, but also to the process of manufacture having interfered with its emulsification, so that it is present in the form of butter rather than in that of cream, which makes it less easy to digest.

Varieties of "Cow and Gate Milk" and "Glaxo" can now be obtained from which a quarter or a half of the cream has been removed. These forms are more generally useful than the kinds with full cream; for, in feeding infants with dried (as with fresh) cow's milk, more protein and less cream should be allowed than are found in breast-milk, owing to the different characters of these substances in the two forms of milk. Dried milk is rather an expensive form of food.

Ass's Milk.—The chief characteristics of ass's milk are its low percentage of fat and of protein, and the fact that a considerable proportion of the protein is in the form of lactalbumin, so that the curd which it forms resembles that of human milk in its physical qualities. Its composition varies considerably; it may be given approximately as follows:—

Average Composition of Ass's Milk (Still).

Fat	1.0	Lactalbumin	0.8
Milk Sugar	5.5	Salts	0.4
Casein	1.0	Water	91.3

While its low nutritive qualities make it an unsatisfactory food to give for any length of time to an older infant, ass's milk is extremely useful for the temporary treatment of young babies who are unable to digest cow's milk in any form, and also in various forms of indigestion. It is, for example, valuable in some cases of coeliac disease. It should be given undiluted and unboiled, and it may be safely used raw, as the ass is practically immune to tuberculosis. Unless a low percentage

of fat is desired, the cream may be supplemented by cod-liver oil emulsion, or, in some cases, by ordinary cream. If it proves too laxative, citrate of soda or liq. calc. sacch. (10 minims to 1 oz.) may be added.

Goat's Milk.—Goat's milk is not very different in composition from that of the cow :—

Average Composition of Goat's Milk (König).

Fat	4.07	Salts	0.85
Milk Sugar	4.63	Water	86.88
Protein (mostly casein)	3.76		

The fat droplets in it are said to be smaller than those in cow's milk; but the casein coagulates quickly and forms compact curds. When it is largely used in hot climates, as in some parts of India and on the Mediterranean, it has the disadvantage that it may communicate the infection of undulant (Malta) fever. Goats, however, are very rarely affected by tuberculosis.

Proprietary Infant Foods.

There is a large variety of "infant foods" on the market; and, although they are not in themselves of much value in the feeding of babies under ordinary circumstances, they are so largely made use of by the public that the medical practitioner must know something about the composition of the most popular of them. It is generally claimed that they are "perfect substitutes for mothers' milk." One has only to glance at their analyses—even those published by the makers themselves—to see that this claim is not justified, and that their composition and characters are very different from those of human milk. They have the further disadvantages that, as they yield large profits to their makers and are much advertised, they are very expensive; and that they are often accompanied by statements which are calculated to mislead the ignorant mothers who buy them, because they give an exaggerated estimate of their usefulness and universal applicability to all babies.

The great majority of proprietary infant foods are, from their composition, seriously deficient in accessory food factors, which goes far to explain the flabbiness and subnormal resistance to disease which infants fed on them frequently show.

The accompanying Table (pp. 188-192), which was originally

prepared by the late Dr E. Bronson, shows the composition of the "infant foods" which are most largely used in this country. It includes also that of some of the dried milks, as they are sometimes advertised as "infant foods."

The different proprietary foods may be roughly divided into milk foods, malted foods, and farinaceous foods.

(1) The **Milk Foods**, which include some forms of dried and condensed milk, are made from cow's milk, with or without various alterations and additions. They only need the addition of water to prepare them for use. Most of them have too little cream and too much sugar. Examples of this group are Allenbury's No. 1 and No. 2 Foods, Carnrick's Soluble Food, Horlick's Malted Milk, and Milo Food. All of them have the advantage, from the point of view of digestion, that the casein they contain is in an easily digested state and clots loosely, if at all, with rennet, but their low percentage of fat renders them unfit for prolonged use.

(2) **Malted Foods** consist of cereals, the starch of which has been changed more or less completely into soluble forms of carbohydrate. In some of these the dextrinised cereal is combined with condensed or dried milk. To the others milk has to be added; for, if mixed with water only, they are altogether deficient in fat, protein, and mineral salts. They may be divided into two classes:—

- (a) Those in which the dextrinisation of the starch has been carried out completely before the food is offered for sale. This includes Mellin's and Cheltine Foods and Hovis Babies' Food No. 1. Completely malted foods are sometimes useful in the treatment of certain food disorders in which dextrose and maltose are more easily digested than milk sugar.
- (b) Those containing starch combined with malt and pancreatic ferment, so that it is partially converted during the process of preparation of the food, *e.g.*, Benger's Food, Moseley's Food, Savory and Moore's Food, and Allenbury's No. 3 Food.

When the malted foods are added to cow's milk they sometimes make it more easily digested, probably in the same way as barley-water does; and they are therefore useful in certain cases as a temporary addition to the diet after the sixth month.

TABLE OF THE COMPOSITION OF INFANT FOODS.

GROUP I.—MILK FOODS.

SUB-GROUP A.—DRIED MILKS.

The fat in dried milks is usually in an unemulsified form. Water only to be added.

	Water.	Salts.	Fat.	Protein.	Carbohydrate.	When diluted, 1 drm. ¹ by weight to 1 oz. of water.			REMARKS.
						Fat.	Protein.	Carbo- hydrate.	
Cow & Gate (Full cream) ³	2.4	5.6	25.5	25.0	35.2	3.2	3.1	4.4	An easily digested dried milk, but unsatisfactory for continued feeding because of the low fat content. One level tablespoon = 16 calories. A dried whole-milk prepared by the "spray process." A milk in which the fat, sugar, and protein are brought to proportions as in human milk and then dried. A dried milk with added farina. Dried whey. Very low fat content—recommended only for premature infants.
" (Half cream).	2.4	6.5	15.5	28.3	41.0	1.7	3.2	4.5	
" (Separated)	3.1	7.4	0.7	33.0	49.5	0.1	3.3	4.9	
Glaxo (Full cream) ²	3.25	2.7	5.25	
" (Three-quarter cream)	2.5	3.37	5.5	
" (Half cream).	1.75	3.7	5.8	
Dryco or Honour Brand ³	3.0	7.0	12.0	34.0	44.0	1.5	4.0	5.5	
Trumilk ⁴	3.8	6.0	27.4	25.6	37.2	3.4	3.2	4.65	
Trufood (humanised) ³	1.48	5.48	28.97	11.82	52.25	3.45	1.4	6.25	
Berina ³	3.04	3.97	17.30	19.74	55.95	2.8	3.29	9.82	
Secway ³	1.0	9.0	1.0	13.0	76.0	

SUB-GROUP B.—SWEETENED CONDENSED MILKS.

The low protein and lactose in these foods indicate the small amount of milk solids in their composition. As the analyses indicate they are inadequate as foods. One part is added to five parts of water.

Borden's Eagle Brand ⁵	25.66	1.78	9.61	8.01	54.94	1.6	1.3	9.15	Of the total carbohydrates, 42.81 per cent. is cane-sugar and 12.03 per cent. lactose.
Milkmaid Brand ⁶	23.7	2.3	11.0	9.7	53.3	1.8	1.6	8.8	Of the total carbohydrates, 38.7 per cent. is cane-sugar and 14.6 per cent. lactose.
Nestlé's ⁶	22.8	1.6	13.7	9.7	52.2	2.28	1.6	8.7	Of the total carbohydrates, 37.2 per cent. is cane-sugar and 15 per cent. lactose.

SUB-GROUP C.—UNSWEETENED CONDENSED OR EVAPORATED MILKS.

With the water replaced, these foods are supposed to have the composition of cow's milk. One part is added to two parts of milk.

Borden's Evaporated Milk ³	73.27	1.43	8.1	7.15	10.05
Ideal ⁶ . . .	62.0	1.3	12.4	8.3	16.0	4.1	2.7	5.3	
Hollandia ⁶ . . .	57.0	3.4	9.8	11.3	18.5	3.26	3.76	6.1	Because the protein is disproportionately low, this analysis indicates that extra cream and sugar have been added so as to resemble the mother's milk more nearly. The analysis indicates the addition of a small amount of sugar.

GROUP II.—MILK AND MALTED FOODS.⁷

This Group consists more or less completely of malted starch combined with dried milk.

SUB-GROUP A.—NO INSOLUBLE CARBOHYDRATES.

This Sub-Group has no insoluble carbohydrates. Water only to be added in the proportion of one oz. by weight to one drm. of the food.

Allenbury's No. 1 ⁶ .	5.7	3.75	18.5	9.7	62.3	2.3	1.09	7.78	Defective in protein, a part of which is of vegetable origin. This food is especially defective in the accessory substances which protect against scurvy.
Allenbury's No. 2 ⁶ .	3.9	3.5	17.6	9.2	66.8	1.9	1.15	8.25	Contains some malted flour. Even more of the protein than in No. 1 is of vegetable origin.
Horlick's Malted Milk ⁶ .	3.7	2.7	9.0	13.8	70.8	1.1	1.47	8.85	Dried milk and malted wheat flour. Defective both in fat and protein, a part of which is of vegetable origin.
Neaves' Malted Food ⁶ .	2.4	4.5	26.0	20.0	47.1	3.25	2.5	5.88	The analysis indicates a fairly satisfactory food, a dried milk with additional carbohydrate as maltose and lactose.

TABLE OF THE COMPOSITION OF INFANT FOODS—continued.

GROUP II.—MILK AND MALTED FOODS—continued.

SUB-GROUP B.—CONTAINING UNALTERED STARCH.

This Sub-Group, to which water only is to be added, gives a very unbalanced diet, suitable only for the most temporary use when a carbohydrate food is desired.

	Water.	Salts.	Fat.	Protein.	Carbohydrate.	When diluted, 1 drm. ¹ by weight to 1 oz. of water.			REMARKS.
						Fat.	Protein.	Carbo- hydrate.	
Carnrick's Soluble Food ⁶	5.3	2.2	2.5	13.6	76.2	0.3	1.7	9.5	Dried skimmed milk, 37.5 per cent.; partly malted wheat flour, 37.5 per cent.; and lactose, 25 per cent.
Milo Food ⁶ . . .	3.6	1.9	5.2	14.0	75.3	0.6	1.7	9.4	Nestlé's Food has practically the same composition. Insoluble carbohydrate, 15.39 per cent.; soluble carbohydrate, 58.93 per cent., of which 25 per cent. is cane-sugar, 27 per cent. maltose and dextrin, the remainder lactose.

GROUP III.—FOODS MAINLY CARBOHYDRATE.⁸

SUB-GROUP A.—SOLUBLE CARBOHYDRATES ONLY.

As the analyses show, these foods offer no advantages over dextri-maltose purchased as such. Used with milk, they supply sugar in a very expensive form. The excess of salts added may be an additional disadvantage.

Cheltine Maltose Food ⁶ .	4.6	2.25	0.27	5.3	87.6	neg- ligible.	0.66	10.95	A malted cereal food containing no unaltered starch.
Mellin's Food ⁶ . . .	6.3	3.8	trace	7.9	82.0	"	0.98	10.25	Essentially a preparation of dextrin and maltose in the proportion of 1 to 3. Gluten cells are

present. This fact, together with absence of lactose, indicates a protein of vegetable origin, *i.e.*, from the malted grain. Potassium bicarbonate added. Caloric value = 105 to the ounce by weight.
A fully malted food. Carbohydrates entirely in the form of maltose and dextrin.

SUB-GROUP B.—CARBOHYDRATES PARTLY INSOLUBLE (STARCH).

This Sub-Group, also to be used with milk, are useful though expensive and unnecessary additions to a milk formula when one desires to start starch-feeding. Well-baked wheat flour is equally satisfactory, especially when prepared with malt.

Allenbury's Malted Food ⁶	6.5	0.5	1.0	9.2	82.8	0.12	1.15	10.35	A mixture of wheat flour and malt. It contains unaltered starch when prepared for use.
Benger's Food ⁶ . .	8.3	0.8	1.2	10.2	79.5	0.15	1.27	9.8	A mixture of wheat flour and pancreatic extract. Both starch and proteins are partially digested in its preparation.
Moseley's Food ⁶ . .	10.8	0.94	0.92	11.0	76.4	0.11	1.7	9.55	When well prepared, all starch is converted.
Savory & Moore's Food ⁶	4.5	0.6	1.4	10.3	83.2	0.17	1.3	10.4	A mixture of wheat flour and malt. Nearly all the starch is converted.

SUB-GROUP C.—ESSENTIALLY STARCH FOODS.

These Foods are advertised to be used with milk. In analysis they are similar to wheat flour, with a varying proportion of the starch changed by baking to dextrin and maltose. If the mixtures are "prepared according to directions," most of them contain too much starch and too little milk.

Wheat Flour ⁹	1.0	11.4	75.1	0.12	1.4	8.39	Raw arrowroot starch and cooked cereal starch.
Eskay's Food ³ . .	3.06	1.34	1.2	6.56	87.8	0.15	0.82	10.97	Contains a small percentage of whole egg. Insoluble carbohydrates about 21.21 per cent.
Frame Food Diet ⁶ . .	5.0	1.0	1.2	13.4	79.4	0.15	1.67	9.9	Essentially a baked flour to which cane-sugar has been added.
Neave's Food ⁶ . .	6.5	1.6	1.0	10.5	80.4	0.12	1.3	10.05	Baked flour. The percentage of unconverted starch varies.

TABLE OF THE COMPOSITION OF INFANT FOODS—continued.
GROUP III.—FOODS MAINLY CARBOHYDRATE—continued.
SUB-GROUP C.—ESSENTIALLY STARCH FOODS—continued.

	Water.	Salts.	Fat.	Protein.	Carbohydrate.	When diluted, 1 drm. ¹ by weight to 1 oz. of water.			REMARKS.
						Fat.	Protein.	Carbo- hydrate.	
Ridge's Food ⁶	7.9	0.7	1.0	9.2	81.2	0.12	1.15	10.15	Baked flour, in one analysis containing only about 3 per cent. of soluble carbohydrates. The various analyses differ widely in this respect. Barley need not be patented to be a useful cereal. Ground oats from which the husk has been removed. Rich in protein and mineral matter. A fine oat-flour. Somewhat inferior in nutritive value to "groats." Starch unaltered. Contains about 7½ per cent. of starch. To be made up with milk. Contains starch when prepared for use.
Robinson's Patent Barley ⁶	10.1	1.9	0.9	5.1	82.0	0.1	0.6	10.2	
Robinson's Groats ⁶	10.4	1.7	1.6	11.3	75.0	
Scott's Oat Flour ⁶	5.8	1.3	5.0	9.7	78.2	
Hovis Babies' Food No. 2 ⁶	2.4	1.7	0.1	5.7	90.1	
Cheltine Infants' Food ⁶	7.2	1.83	3.92	16.2	71.0	
Sister Laura's Food ⁶	1.18	1.10	1.78	16.52	79.42	3.96	3.89	7.67	It is recommended to add 1 teaspoonful to 5 oz. of undiluted milk. It is claimed that its chief value consists in its helping the digestion of the milk.

¹ Two level teaspoonfuls weigh about 1 drm.

² Still, *Common Disorders of Childhood*.

³ Manufacturer's Analysis.

⁴ Mendel and Osborne, "Feeding Experiments," *Carnegie Institute Rep.*, 156.

⁵ Holt, *Diseases of Infancy and Childhood*.

⁶ R. Hutchison, *Food and the Principles of Dietetics*.

⁷ As the dilution of 1 drm. to 1 oz. of water is used with these dried foods, comparison with dried milk may be made.

⁸ Two level teaspoonfuls approximate 1 drm. by weight, hence 1 teaspoonful to the ounce of milk adds about 5 per cent. of carbohydrate.

⁹ Morse and Talbot, *Diseases of Nutrition and Infant Feeding*.

(3) **Farinaceous Foods** (Group III., Sub-Group C in the Table) are composed of wheat or some other cereal, the starch of which has not been acted on by malt or pancreatic ferment. We have already seen that starch is not, as a rule, a suitable food for babies before the beginning of dentition. These foods form quite a suitable addition to the diet of older infants, but they are more expensive, and in no respect better than such ordinary foods as wheat, barley, or oat-flour, or even than rice or rusks. Ordinary forms of flour can be easily malted at home, if necessary, by the use of a home-made infusion of crushed malt such as Sir Wm. Roberts recommended (Appendix F).

A study of the Table will show that, when prepared according to the directions, they are all deficient in composition in various particulars. It must also be pointed out that, although the chemical composition of some of them seems at first sight satisfactory, they are very inferior to human milk in other ways.¹ Some of these may be stated as follows:—

(1) **Fat Content.**—Nearly all of them contain too little fat; and in some the fat, though sufficient in quantity, has had its emulsification destroyed in the process of manufacture so that it is more difficult to digest than that of fresh milk.

(2) **Protein Content.**—Many of the foods contain too little protein. In many also the nitrogenous constituent is a vegetable albumin which is not equivalent to the protein of milk, as only about 30 per cent. of it can be utilised by the baby. (See p. 131.)

(3) **Carbohydrate Content.**—Most of them contain far too large an amount of carbohydrate; and many are unsuitable for young infants because so much of it is unaltered starch.

(4) **Antiscorbutic Food Factors.**—All are more or less deficient in antiscorbutic qualities. Most of the infantile scurvy we see in this country is in infants who have been fed for many months on proprietary foods.

¹ Sister Laura's food differs essentially from most of the others in the claim that its chief value lies, not in the additional nourishment it supplies, but in its making the milk to which it is added more digestible. There seems to be some ground for this claim. The reasons why many children thrive on it are apparently the relatively small amount of the food added to each bottle, and its being *prepared with full milk*.

The Feeding-Bottle.

The bottle used in feeding the infant should be as simple in form as possible—a plain flask with no angles in it. It should be capable of holding from 8 to 10 oz., and it is sometimes an advantage to have it graduated in half-ounces. It should have no cork or tube connected with it, and only a plain conical rubber nipple fitting over its neck that can be easily turned inside out and scrubbed with a nail-brush. The opening in the nipple should vary in size according to the strength of the child. Generally speaking, it should be large enough to allow the milk to drop through it rapidly when the bottle is turned upside down, but not so large as to let it run from it in a stream. A good form of bottle is that which has an opening at both ends, so that a stream of water can be run through it for cleansing purposes.

Any bottles the nipples of which are situated at the end of a long rubber tube, with a glass tube hanging inside, are altogether objectionable. It is practically impossible to keep them clean, and they encourage ways of feeding the baby which are bad for him, however much they may seem to save the mother trouble.

It is well to provide at least two bottles and two nipples, so that they can be used alternately. Far the best plan, however, is to have a separate bottle for each feed. When this is done, all the milk for the day can be modified and sterilised at the same time, and the bottles filled, stoppered with cotton wool, and set in a cool place. They are then safe from contamination and ready for use when required.

The Use of the Bottle.

When the mother or nurse gives the bottle, she should sit down, take the baby on her knee, and pay as much attention to the process as she would to that of suckling. The food should be given at about the temperature of the body, and the child allowed fifteen or twenty minutes at most for his meal. Regularity in the times of the meals is even more important than in the case of breast-feeding, and the intervals should be the same.

When the meal is over, the bottle and nipple must at once be thoroughly cleansed with soap and hot water. It is advisable,

and in hot weather necessary, that all the bottles for the day should be sterilised by boiling at the time the day's food is being prepared. No milk which has been for any length of time in the bottle should be given; and if any drops have dried on the glass inside they must be carefully removed with soda. The rubber teats must be frequently renewed, as they are apt to develop cracks, and then it is impossible to keep them clean.

The importance of keeping the feeding-bottle free from sourness cannot be over-estimated. A single bottle of contaminated milk may start a serious, or even a fatal attack of diarrhœa. The habit, which some nurses have, of putting the rubber teat to their own lips before giving it to the baby is *very dangerous* and should never be permitted.

The child should never be allowed to suck the nipple of an empty bottle; and the constant sucking of an imperforate rubber "comforter" is a sign of bad nursing and a bad habit. It must, however, be admitted that, in exceptional cases, the temporary use of a contrivance of this kind may be of real value in soothing a dangerously ill child whose condition makes it imperative that he should be kept from crying. If it is ever allowed, the teat must be frequently sterilised by boiling and never let fall on the floor while being used.

Food, other than Milk, Suitable during the First Year.

When the baby is six or seven months old his first teeth usually make their appearance; and, when several of these are through the gum, this is a natural indication that the digestion is becoming fit for more complicated food. The child also often at this time shows signs of being less satisfied with his exclusively milk diet, and gains weight less steadily. It is then usually advisable to begin some form of starchy food, though, if the child is growing quite satisfactorily and is contented, it may be just as well to go on with milk only for another month or two, especially if the teeth are late in coming. When, however, in a healthy child the teeth are very late in appearing, this should not of itself be considered a sufficient reason for deferring indefinitely the use of farinaceous food. When the baby is nine or ten months old he should have five meals a day. These should consist mainly

of milk, preferably undiluted, but to the first and fourth some suitable farinaceous food should be added. Oat-flour does very well, or barley or wheat flour, barley jelly or bread jelly (see Appendix F), or one of the proprietary foods.

It is also well about the tenth month to add some additional nitrogenous food to the midday meal, such as clear soup, beef tea or chicken tea, or to give half the yolk of a raw or lightly boiled egg beaten up with the milk. Egg yolk possesses a greater degree of concentration of the anti-rachitic factor than any other foodstuff, and is rich in iron (8.6 mgrm. per cent.). The following analyses are of interest in this connection:—

Ordinary Beef Tea (Cheadle).	White of Egg (König).	Yolk of Egg (König).
Water . . . 96.31	Water . . . 85.50	Water . . . 51.03
Fat . . . 0.00	Fat . . . 0.25	Fat . . . 31.39
Protein . . . 0.82	Protein . . . 12.87	Protein . . . 16.12
Extractives . . 2.09	Free Extractives 0.77	Free Extractives 0.48
Salts . . . 0.78	Salts . . . 0.61	Salts . . . 1.10

The appearance of the teeth affords another important indication as to the infant's feeding, besides calling for the addition of starch to the diet. It points to the need of the child having some form of food that will give the new teeth something to do and train him in the use of his jaws for other purposes than mere sucking (p. 55). It is therefore a natural and proper thing now to give him a crust or piece of baked bread to chew, or to let him gnaw a chicken or chop bone occasionally.

It is also wholesome for the child, at this stage, to have a little fruit juice (orange or lemon, from $\frac{1}{2}$ to 1 oz. in the day); and to let him further exercise his teeth and jaws in getting it. This may be done by giving him a section of a ripe orange to chew, after the pips have been removed. Stewed or baked apple may also be given; and it is a good thing to dilute the milk with water in which vegetables have been boiled in order to increase the salt intake. A mistake which is often made is to give a great excess of starchy food in place of milk. This is a fruitful source of indigestion and anæmia. Another mistake is to continue to add sugar to the child's milk and to his farinaceous food. The starch is meant to take the place of

the sugar to a large extent in the diet; and the continued sweetening of the food is injurious, because it lessens the child's natural relish for the taste of cereal and fatty foods, and creates an unnatural craving for sugar. Pap made of boiled bread and milk to which sugar has been added is a very pernicious form of food for babies. Even at this age it is well to give him a little baked bread (p. 58) with salt butter.

Food during the Second Year.

After the end of the first year the infant's midday meal should be increased. In addition to what has been already mentioned, he may have, in moderation, such things as potato, rusk, or bread-crumbs with gravy, such easily digested vegetables as spinach, cauliflower, asparagus, or vegetable marrow, a lightly boiled egg, custard pudding, curds (junket), well-boiled porridge, or any plain farinaceous pudding, without the addition of sugar. Baked bread or toast with salt butter, in gradually increasing amounts, should form a regular part of his diet. Oatmeal porridge is often objected to by dentists and physicians; but, though it disagrees with some children and should not therefore be given to them, it would be difficult to deny that, when well made of good meal and well salted, it is for the majority of children a most wholesome as well as an appetising food. When made of inferior meal without salt, and eaten with sugar, as we often get it in England, it well deserves the bad things said about it. When we consider that the solid food given to a child in his second year requires certain alterations before he can digest it, we must remember also that Nature has made provision for this, and that the child is bound to suffer, if he is not allowed to make full use of the organs with which he has been supplied for this purpose. In other words, we must bear in mind that the teeth, jaws, and muscles of mastication, on the natural development of which the future health will to a large extent depend, should have a normal amount of exercise at this time of life. A diet which gives no exercise to the jaws and discourages the free secretion of saliva usually results in the jaws becoming undergrown and deformed, and consequently predisposes to crowding and decay of the permanent teeth. This question is treated in greater detail on p. 57.

In the second year, milk should still form a large part of the child's diet. It need not usually be diluted and no sugar should be added to it; for he is getting plenty of carbohydrate in the form of starch, and it is good for him now to have it in that form. By the fifteenth month, at latest, the bottle should be discontinued and all the milk given from a cup. Thirty ounces of milk in the day are sufficient for a healthy child at this age.

After eighteen months, four meals a day should be given and it will be better for the child not to be wakened for a late meal, but allowed to sleep right on to the morning. A little fish, chicken, rabbit, or butcher meat, fat bacon, or a lightly boiled egg may be given for dinner. The meat should not be minced too finely, as it is most important that the child should learn to chew his food. The amount of potato and pudding is also to be increased, and stewed fruit and vegetables given, as well as baked bread with butter.

Alcohol, tea, and coffee should, of course, form no part of a young child's diet; and pastry, cheese, and sweets are also unsuitable at this age. Little or no water should be given with the food; but it is good for the child to be allowed a drink of water between his meals. Small extra meals, consisting of milk and biscuits, or bread and butter, given on waking, between breakfast and dinner, or the last thing at night, are not good for healthy children.

Diet in Older Children.

Children who have been judiciously fed and wisely trained from early infancy are not likely to give trouble in the matter of diet as they grow older. Their healthy appetites, normal tastes, and sound digestions may be trusted to be helpful guides as to what they ought, and what they ought not, to have. Unfortunately, however, a majority of the children whose feeding we are asked to supervise have already acquired bad habits of eating and abnormal tastes. These by long practice have become a second nature to them, so that it is necessary to explain to their parents the main dietetic rules which should guide them, and even to write out a list of the articles of which the child's meals should consist.

A few general principles may be stated as follows:—

I. Composition of the Meals.—Although an increase of starchy food is natural and beneficial at this stage, we must

guard against the common mistake of giving such food in excess. In many cases loss of appetite, debility, and chronic intestinal indigestion are due to errors of this kind.

It is important that the growing child should have plenty of animal protein; but this also must not be given in too large an amount. Many children have a craving for meat. In some hospitals, owing to this, a practice has arisen of giving the pudding course of the dinner before the meat, because it is found that, when the meat is given first, the children cry for more and refuse the pudding. Perhaps in the case of poor children this may be an indication that they need more animal food than they have been getting.

Sufficient fatty food is also important, and the bad habit of leaving the fat of the meat must be checked from the beginning, and butter also freely allowed. A taste for vegetables should always be encouraged, but an excess of potatoes is to be avoided. Fruit is very wholesome for healthy children, although it may disagree with those whose intestinal digestion has been weakened by too much starchy food. Bananas if unripe are indigestible, but if ripe, and especially if cooked, they usually agree quite well if the digestion is normal. Ripe apples are the most wholesome form of fruit for children. With a few exceptions, all fruit is best given cooked to delicate children. Sweets in moderation and at proper times are natural and wholesome, but the great danger to the teeth from their injudicious use is never to be forgotten (p. 58).

The child's taste for the ordinary food which is good for him needs to be cultivated.

2. Form of the Meals.—The child must be taught to eat slowly and to chew his food well. With this in view, the natural taste of it should not be interfered with; and a fair proportion of his starchy food should be given in a hard, dry form and not, as is often done, in a pulpy, relatively tasteless or over-sweetened mass which discourages chewing.

Not only should the meals contain the food in proper forms, but they must also be arranged physiologically; and it is an advantage if they do not end with sticky carbohydrates which lodge about the teeth, or with sweets. The ideal thing is that such things as "milk puddings," bread and jam, cakes, biscuits and oatcakes should be followed by fresh fruit, such as a ripe apple thoroughly chewed, to scour the teeth and incite the

secretion of active saliva. Some hard form of carbohydrate such as rusk or baked bread is also useful in this way; and it is always advisable that, at the end of the meal, the mouth should be rinsed out with plain water, or with a suitable mouth-wash (p. 60). The essential point for the preservation of the teeth is that the mouth should be left clean after eating.

The child should not take much water along with his food; but in warm weather and after exertion it is good for him to drink freely between meals.

It may be said in general that the child should be allowed a mixed general diet, with as great a variety as possible, so that the various salts and vitamins be provided. The eating of green vegetables such as greens and spinach is to be encouraged. The only articles to be avoided are stews and spiced dishes. Freshly prepared dishes are to be preferred to tinned articles, although tinned tomatoes are useful in that they retain their anti-scorbutic powers.

Many parents are obsessed regarding the amount their children eat, and frequently complain that the child does not eat enough. It is often very difficult to get exact details on this matter, but it should be remembered that the faecal output is as a rule a good indication of the amount of food taken. It may also be remarked that most healthy children take a good breakfast and midday meal but a comparatively small supper or tea; they seem to require their food while they are expending energy. If the child is growing quickly he may develop an inordinate appetite; this is often noticeable prior to puberty.

The following may be taken as an example of a suitable dietary for a healthy child:—

Breakfast (at 8 or 8.30 A.M.)—Porridge and milk, a lightly boiled egg, fish, or bacon, baked bread or well-done toast with salt butter and milk; finishing with a ripe apple.

Dinner (at 1 or 1.30 P.M.)—

A. (1st Course).—Pea soup, lentil soup, or potato soup.

B. (2nd Course).—Meat, fish, chicken, or rabbit. Potato and vegetables.

C. (3rd Course).—Pudding and stewed fruit or fresh fruit in season.

It is a frequent experience that a child will not take all three courses. Course *B* should invariably be given. On certain days

give *A* and *B*, and on other days *B* and *C*. Course *A* should only consist of really nutritious soups and those mentioned are the ones recommended.

Tea (at 4.30 or 5 P.M.)—Rusks, toast or baked bread with butter or jelly, or plain cake, milk alone or merely flavoured with cocoa or tea, a raw apple.

Supper (if still hungry)—Before or after going to bed a drink of warm milk without any starchy food and no sweets.

The following is an example of a kind of diet often given, which is calculated to leave the mouth in such a state as to encourage to the utmost the growth of acid-forming bacteria about the teeth:—

On waking—Milk and a biscuit.

Breakfast—Porridge and milk, or boiled bread and milk, soft bread and jam.

Lunch (a few hours later)—Milk and a sweet biscuit.

Dinner — Mashed potatoes and gravy, minced meat, farinaceous pudding and milk.

Tea — Boiled bread and milk, or bread and jam, cake, and cocoa.

Supper (on going to bed)—A glass of milk and a biscuit, and a small piece of chocolate or other sweet.

When the diet of the children in an institution has to be arranged, it is necessary to go fully into the details of the composition of the meals. The caloric value of the food given, the relation of its protein and non-protein elements, that between its carbohydrates and fats, the amounts and characters of its mineral salts, and the quantity of liquids taken have all to be considered. It must also be seen that the diet contains sufficient vitamins and extractives; and the variety of the meals, their taste, cooking, digestibility, absorbability, and other physical properties have to be taken into account, as well as their cost.¹

¹ Much interesting and practical information on these matters will be found in a small book on *Food Values*, by Margaret M'Killop, M.A., London (Routledge), 1916; and in *The Physiological Feeding of Children*, by Dr Eric Pritchard, London (Kimpton), 1919.

CHAPTER IX

THE FOOD AND NUTRITIONAL DISORDERS OF INFANCY AND OTHER FORMS OF DIGESTIVE DISTURBANCE

IT may be in consequence of the fact that digestion and absorption during infancy are working at their maximum that they are so easily upset, or it may be because of an inherent weakness of the infantile digestive system, but whatever the reason, it is a fact that in almost any diseased process at this age some disorder of the gastro-intestinal system, with involvement of nutrition, is apparent. This may take the form of vomiting, diarrhœa, failure to gain in weight, or loss of weight. These symptoms dominate the clinical picture so much at times that there would seem to be little question that they are primarily of gastro-intestinal origin, and definitely associated with the type of the food which the child is receiving. There is no doubt of course that in a certain number of instances the gastro-intestinal symptoms are due to the type of food ingested. More frequently, however, they are in consequence of the food being contaminated by the products of decomposition, or some pathogenic organism, than to the direct action of the proximate principles themselves. But most frequently of all, the gastro-intestinal manifestations are the result of disease in some other part of the system. In these circumstances they are said to be *parenteral* in origin. To approach nutritional disease in the infant unless from this broad outlook will lead to the most serious of errors. For the physician the importance of the above conception is that a most thorough physical examination in search of some focus of disease other than in the gastro-intestinal tract must invariably be made, special attention being directed to the ears and urine, and that it is only when no such focus can be found that any idea of some so-called food-disorder should be entertained.

There have been many classifications of nutritional disease, and, as has been the case with disease in general, the outlook

on the cause and nature has been reflected in the particular advance in medical science which prevailed at the time. At first it was the custom to name the condition according to the most prominent symptom, *e.g.*, atrophy or marasmus, when emaciation was the dominant feature of the illness, or gastritis, gastro-enteritis or cholera infantum, when vomiting and diarrhoea predominated. Later on, in the days when morbid anatomy held the field and was bringing order out of chaos in many departments of medicine, an attempt was made to classify nutritional diseases according to the pathological lesions found after death, as *e.g.*, gastro-enteritis, follicular enteritis, and ulcerative enteritis. This system, however, proved unsatisfactory, not only because it was exceedingly difficult, indeed impossible, to identify the particular type of lesion during life, but also because many of the most severe examples presented no appreciable lesion after death.

When bacteriology was elucidating the cause of many diseased processes it was only natural that gastro-intestinal disease should be similarly investigated, and in fact Escherich made an attempt to differentiate the various clinical pictures according to the bacterial flora of the gut. This method, however, for the generality of nutritional or intestinal disease, proved even less satisfactory than that of morbid anatomy, although it is being resuscitated by Brown of Toronto and his co-workers. We know that a sterile intestinal canal is incompatible with life, and that normally within the gut there are many organisms which possess pathogenic powers, but that so long as they remain within the confines of the intestine, *i.e.*, in reality outside the body, they serve a beneficial purpose in counteracting the growth of intruders, and in aiding digestion. But to decide when the various coliform types and streptococci have reached a pathological proportion, or have assumed a special toxicity, has so far proved impossible. If, however, some organism which is not a normal inhabitant of the gut is detected, *e.g.*, one of the dysentery group, one of the typhoid group, or the tubercle bacillus, then bacteriology is of the greatest service. It is on the basis of the bacteriological findings that we have learned to recognise ileo-colitis or dysentery and typhoid fever as specific infections, two diseases which belong to the group we are at present discussing, and which are not uncommon during later infancy and early childhood.

At a still later date, when metabolic processes were arousing interest, Czerny¹ suggested that at least certain types of nutritional disease were due to the abnormal reactions of the child, or of its gastro-intestinal system, to the proximate principles of the food. Until Czerny's work the only constituent of the diet which was considered harmful was the protein. Because these nutritional or digestive disturbances were most common in the bottle-fed child, and because the greatest difference between human and cow's milk seemed to be in the protein content, medical opinion was obsessed with this factor, and not only were many digestive disorders during infancy considered due to this element, but all modification of cow's milk for infant feeding concerned itself with the rectification of this difference. Czerny was one of the first to exonerate protein from having any harmful influence, and suggested that it was rather the fat and carbohydrate which were responsible for any abnormal results which developed on feeding with cow's milk. He described two definite clinical pictures: (a) *Milchnährschaden*, the result of feeding with concentrated milk mixtures and in which he saw the action of the fat; and (b) *Mehlnährschaden*, the result of feeding with dilute milk mixtures containing an excess of added carbohydrate. He also postulated that these conditions occurred not only in children when fed with excessive amounts of fat or carbohydrate but that certain children, on account of a lowered tolerance to these substances, either in consequence of disease or in virtue of some inherent weakness, became similarly affected when given fat and carbohydrate even in normal amounts. This was the first suggestion of the conception of the make-up of the child being a factor in the cause of nutritional disease.

Finkelstein, while admitting that there was much truth in Czerny's thesis, drew attention to another feature of the diet (cow's milk) which, although not itself determining the mischief, certainly predisposed to it. Imbued with the teachings of Jacques Loeb on the influence of inorganic salts on the development of the lower forms of animal life, Finkelstein suggested that some of the digestive disturbances might be caused through the action of the salts in the milk. As the milk of each species of mammal has a particular salt content, it seemed reasonable to conclude that the intestinal mucosa would function best

¹ J. Loeb, *Künstliche Parthenogenese*, Leipzig, 1906, and *Studies in General Physiology*, London, 1905, 612.

when the food constituents were suspended in a medium designed by Nature. We have already seen (p. 132) that absorption, or at least the utilisation, of the various food constituents is better in the infant when fed with human milk than with cow's milk, and the well-known whey-exchange experiments of Meyer¹ are further support of Finkelstein's theory. In these experiments cow's milk and human milk were curdled and the curd separated from the whey. The curd of the cow's milk was then mixed with the whey of human milk and the curd of human milk with the whey of cow's milk. These two mixtures were fed to infants and it was found that those fed on cow-curd and human-whey did equally as well as, or even better than, children given breast-milk, whereas those fed on human-curd and cow-whey reacted as if fed on cow's milk. These latter children had undigested motions and showed less satisfactory gain in weight. Since the only radical difference between the two wheys was in the amount and proportion of the various salts, there seemed little doubt that this was the cause of the varying behaviour.

And still more recently, as is not surprising, the suggestion has been made that nutritional disease in infancy may be of the nature of an avitaminosis—a deficiency disease. A deficiency of the growth-promoting factor—fat-soluble A—would certainly interfere with development, and it is not improbable that some of the effects of feeding with dilute milk mixtures and condensed milks, poor in fat and rich in carbohydrates, are due to this cause. We have already drawn attention to the fact that the iron content of the milk is barely sufficient to cover the infant's needs, and hence any diminution of this important element, *e.g.*, by dilution of the milk, will, from the consequent anæmia, interfere with nutrition.^{2, 3}

In clinical practice, however, it is found impossible to bring all cases under any one of the above systems of classification. Nevertheless, there can be no doubt, as Czerny has shown, that diet does affect the state of nutrition, and there is also no doubt that infections far removed from the bowel, *e.g.*, of the ear and throat, interfere with digestion and nutrition. There is, how-

¹ L. F. Meyer, *Ergeb. d. inn. Med. und Kinderheil.*, 1908, i., 331.

² S. V. Telfer, *Glas. Med. Journ.*, 1930, cxiii., 246.

³ Helen M'Kay, "Nutritional Anæmia in Infancy," *M.R.C. Report*, No. 157. 1931.

ever, no difference in the clinical picture whether the emaciation or wasting is due to starvation, to an excess of fat in the diet, or to the effect of a chronic infection like tuberculosis. Nor is there any radical difference in the toxic symptoms and evidences of gastro-intestinal indigestion (vomiting and diarrhœa) with anhydræmia (loss of fluid), whether the exciting toxin is produced locally by decomposing food or pathogenic organisms, or whether the bowel is damaged as the result of toxins produced at a distance and circulating in the blood. Parenteral infections, however, are seldom if ever accompanied by the degree of anhydræmia with the Hippocratic facies so typical of acute gastro-enteritis. There would also seem to be little question that congenital weakness or idiosyncrasy to the effect of the various foodstuffs may play a part. And finally, it must be appreciated that the one type of lesion leads to the other. Toxic action damaging the bowel makes the individual more susceptible to the effects of diet, just as dietary factors render the child more susceptible to infection. Thus the complexity of the question is apparent. In most cases more than one factor is often at play, if not at the same time, then alternately. Hence, in the investigation of any nutritional ailment, the clinical picture and the history of the illness, and of the dietary preceding the onset of the illness, are the sole guides to a correct diagnosis of the underlying cause. And, as it is often impossible to be sure when the reactions ceased to be within the range of physiological variation and had become definitely pathological, a proper perspective of the case can only be obtained by reviewing the history from birth. This is quite an easy undertaking since the patient is only a few months old.

It is thus apparent that with our available knowledge a clinical classification is alone possible. In the main the cases can be divided into two groups according to whether there are or are not toxic symptoms and fever, but there is much overlapping, for, as previously remarked, the one condition may cause the other. In the one group (*intoxication*) the illness is of an acute nature and in the other group (*marasmus*) of a chronic nature. In both there is a failure to gain in weight or a loss of weight, and in both types there may be vomiting and undigested loose stools. The old term *marasmus* embraces those of the chronic non-toxic nature, and under the terms

enteritis or intoxication may be brought together those of an acute and toxic nature.

Finkelstein has suggested a classification in which he divided each of the two main types into a mild and a severe form. The toxic group he designates *dyspepsie* and *intoxikation* respectively and the non-toxic group *bilanzstörung* and *dekomposition*, depending on whether there is merely a failure to gain in weight or a steady and progressive loss of weight. There seems, however, little reason to adopt such an elaborate classification, as the same underlying causes are at play in the different grades of the mischief, and in our consideration of these nutritional abnormalities we will discuss them under two main headings.

MARASMUS, BILANZSTÖRUNG, ATHREPSIA.

This condition is characterised in its earliest stage by a failure to gain in weight. There may also be a certain degree of anorexia. The child becomes pale, soft, and listless. As a rule there is no vomiting at this stage and the motions are normal, but they may be pale and crumbly with a degree of constipation or they may be frequent, small, and dark green in colour. It might seem at first sight hardly appropriate to refer to a simple failure to gain in weight as marasmus, but it is apparent that if this arrest of growth is continued long enough it will lead to a state warranting the designation marasmus, since the child will ultimately be considerably under his normal weight.

It is important to appreciate that this impairment of nutrition will lower the child's vitality and resistance, and render him liable to infection. As the condition gets worse, either in consequence of an intercurrent infection or attack of enteritis, or it may be on account of incorrect feeding, the child definitely loses weight. The loss of weight may become extreme, so that the skin hangs in folds on the emaciated limbs and on the abdomen. The subcutaneous tissues lose their normal elasticity, so that when the skin is pinched up the fold so produced persists unduly and does not, as in the healthy infant, disappear at once on being released. The buttocks are often excoriated from the irritation of the urine and motions, and the skin may be the seat of a septic infection (furunculosis). The temperature

tends to be subnormal, the pulse slow, and the respirations irregular and Cheyne-Stokes in character. The motions may be normal but are frequently undigested, bulky, pale and soapy, or small and dark green in colour and consisting chiefly of mucus (*hunger stool*).

Causes of Marasmus.—1. INANITION is one of the most common causes of this nutritional state. The degree of wasting naturally depends on the degree of underfeeding. If enough is supplied for the vital functions and to repair wear and tear there may simply be a stationary weight, but if insufficient for these purposes is provided then the child will live on his own tissues and thus lose weight. Inanition is met with both in the breast-fed and the bottle-fed child. Seldom is the quantity of breast-milk considered at fault, and, if the breast-fed child is not thriving, it is generally concluded that there is something amiss with the quality, so that instead of practising *allaitement mixte* (p. 167) the child is weaned. It is a golden rule to suspect an insufficient supply of milk in any non-thriving breast-fed child. It is in the bottle-fed child, however, that underfeeding is most frequently encountered. It is truly remarkable on what small amounts of milk a child is sometimes expected to be able to thrive. If any symptoms of dyspepsia (colic or vomiting) appear in the artificially-fed child it is usually deduced that the milk is too strong, and even although the child has in reality been getting an insufficient amount, the quantity offered is further reduced. By dilution of the milk the process of reduction may be carried on till little more than water is being given.

A priori one would not expect vomiting to be a symptom of inanition, and yet such is not infrequently the case. It is difficult to explain this symptom in these circumstances, but it may be that in consequence of the deficient amount of food a less proportion of the hydrochloric acid of the gastric juice is combined, thus leaving an excess in the free state which will exert an irritating action on the gastric mucosa. As a rule the motions are small and infrequent, but they may be frequent; and since they are for the most part composed of intestinal secretions and mucus and have a dark olive green colour (*hunger stool*), care must be taken not to mistake them for diarrhoeal stools and evidence of enteritis and an indication for a reduction in the amount of food. Loss of food from severe

vomiting, as in pyloric stenosis and rumination, produces a similar clinical picture.

The **diagnosis** is made by there being no evidence of disease on complete physical examination, other than the altered state of nutrition, and by a comparison of the amount of food the child is receiving with what he requires. The reader is referred to what has already been written on the food requirements of the infant (p. 175). The only way in which to decide how much milk the breast-fed child is ingesting is to weigh him before and after several feeds. In the case of the bottle-fed child the history of the feeding supplies this information.

The **treatment** simply resolves itself into supplying the requisite amount of food. The method of estimating the amount of food the child requires according to the state of nutrition is described on p. 178. If the child has been underfed for some time over-feeding must be guarded against. From prolonged starvation the gut becomes intolerant and the normal quota might easily induce dyspepsia. It is thus wise not to supply the actual needs right away but to start with less, *e.g.*, three-quarters of that amount, and then, as the child's tolerance improves, to increase the intake to the normal level (Fig. 67).

2. FAT DYSPEPSIA.—There is no doubt that certain children have a reduced tolerance for fat and that feeding with undiluted milk mixtures, milk which is specially rich in fat, or milk to which extra fat or cream is added in the hope that the child will more rapidly increase in weight, interferes with nutrition. This is the condition called *milchnährschaden* by Czerny and *bilanzstörung* by Finkelstein. The child ceases to put on weight although getting an ample caloric intake; he becomes pale, soft and flabby, and the motions are large and crumbly. Although the fat is blamed for producing such a state of matters, and the improvement resulting from feeding with fat-poor mixtures supports this hypothesis, the digestion and absorption of the fat are quite normal. The motions owe their peculiar character to the presence of an excessive amount of insoluble soaps, and it is in this way that the mischief was supposed by Czerny to arise. For the formation of these insoluble soaps an excessive amount of calcium and magnesium is necessary, and hence the absorption of these minerals, and perhaps also of sodium and potassium, is interfered with. In the severer examples there is said to be an abstraction of the

salts from the body. It is this which is supposed to account for the loss of weight, as the water content of the tissues depends on the concentration of the salts. In many cases, however, metabolism experiments have shown that there is no diminished salt retention and hence there must be some other explanation. It has been suggested that the excessive fat in the gut interferes with the absorption of sugar, and Nassau and Schaferstein¹ state that they have by blood-sugar curves shown that this

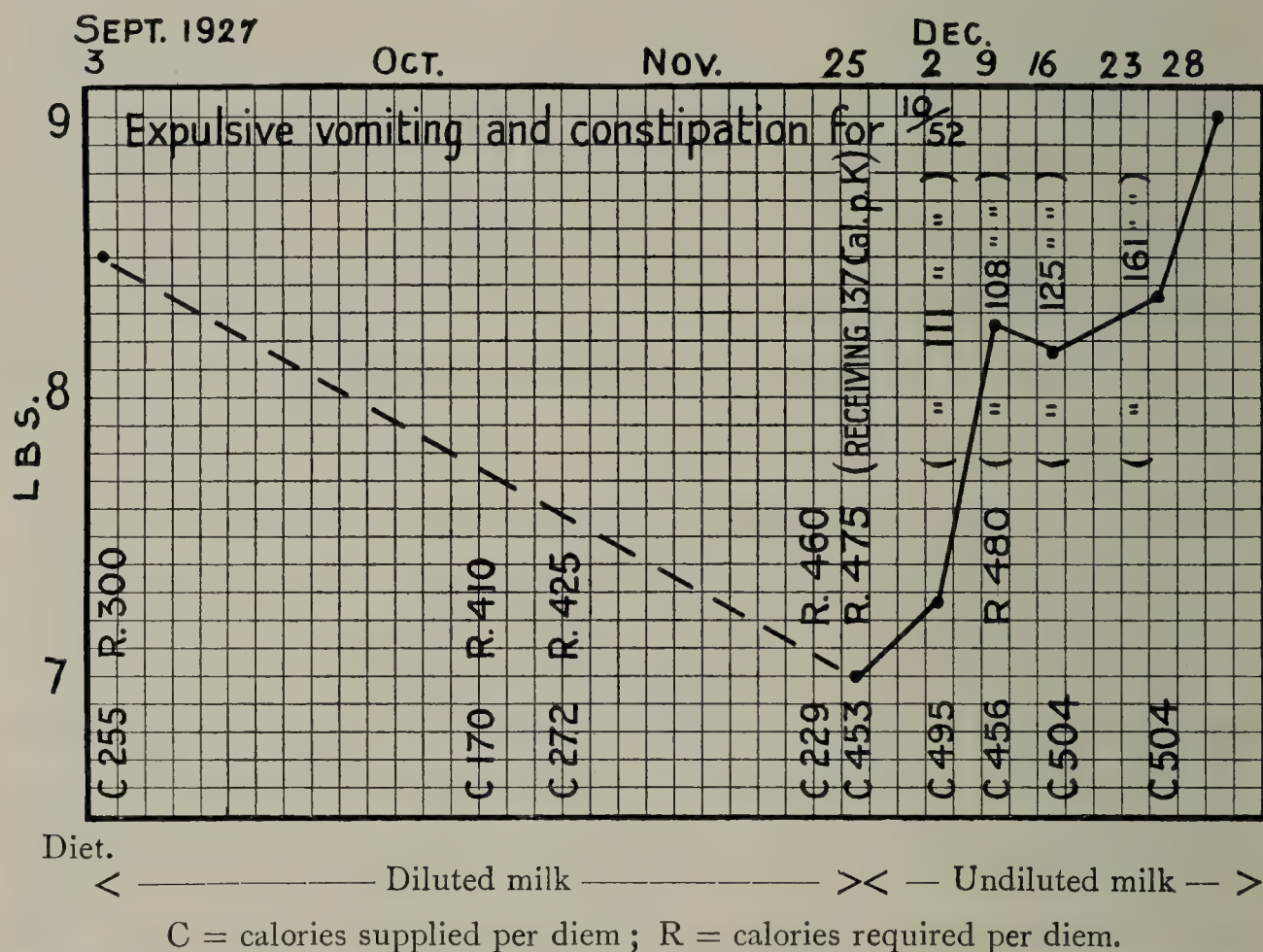


FIG. 67.—Case of Inanition with vomiting, showing recovery after supplying correct amount of food (girl, born 3.9.27).

does happen. However, Muriel Brown,² working at the Children's Hospital, Glasgow, could not detect any difference in the rate of absorption of sugar, whether suspended in milk mixtures containing 1 per cent. or 8 per cent. of fat.

Whatever the explanation of the interference with nutrition during a high fat intake, there is no doubt that it is a definite clinical entity. So frequently did it occur during the use of some dried milks containing a high proportion of cream that the manufacturers now supply brands containing only half the

¹ E. Nassau and S. Schaferstein, *Zeitsch. f. Kinderh.*, 1926, xl., 659.

² Muriel J. Brown, *Quart. Journ. Med.*, 1925, xviii., 175.

amount of cream. These are found to agree much better with young infants.

The **diagnosis** rests on the dietary history (the child having been fed on excessive amounts of undiluted milk or milk to which extra cream has been added), on the presence of large pale and soapy stools, and on the absence of any disease which might interfere with the child's health.

The **treatment** consists in diminishing the fat intake. This can be done by giving diluted milk, but it is better to use undiluted milk from which so much of the fat has been removed, *i.e.*, skimmed milk. It must be remembered in this connection that skim milk as usually on sale is not fit for infant consumption, but dried milks are now on the market containing only one-half of the normal proportion of cream (prescription "Glaxo" and half-cream "Cow and Gate") or even containing no cream—dried skim milk ("Cow and Gate" brand). But the proportion of fat in ordinary milk can be readily reduced in the home by allowing it to stand for some time and then removing the cream which has risen to the surface. If one pint of milk is allowed to stand for four hours the bottom 8 oz. contain fat in the proportion of 1 per cent., and the bottom 12 oz. in the proportion of 2 per cent. The top 12 oz. or 8 oz. may be skimmed off, or the relative complementary amounts of the bottom milk required can be syphoned off. As the removal of the fat reduces considerably the nutritive value of the food extra carbohydrate is usually added. The addition of one dram by weight (two teaspoonfuls) of sugar to every three ounces of 1 per cent. fat milk, or of half a dram to every three ounces of 2 per cent. fat milk, raises the feed to practically a normal nutritive level. It is customary to commence with milk containing 1 per cent. fat, and, as the child improves, gradually increase the proportion of the fat in the feed till a normal diet is being given. This is most conveniently accomplished by gradually diminishing the amount of the supernatant milk which is skimmed off (Fig. 68).

3. PARENTERAL INFECTION.—Infective disease outwith the bowel may also induce marasmus. This may arise in several ways. The disease may interfere with the child's appetite so that a state of inanition results; the diseased process may increase the metabolic rate and cause the child to live in part on his own tissues; or the toxins developed by the infection may damage

the intestinal mucosa and set up the symptoms of enteritis (frequent, loose, and undigested motions), or it may be that the tolerance of the gut is so much lowered that it is the proximate principles of the food (the fat and carbohydrate) which are enabled to exert their baneful influence. In these latter circumstances the train of symptoms described under fat

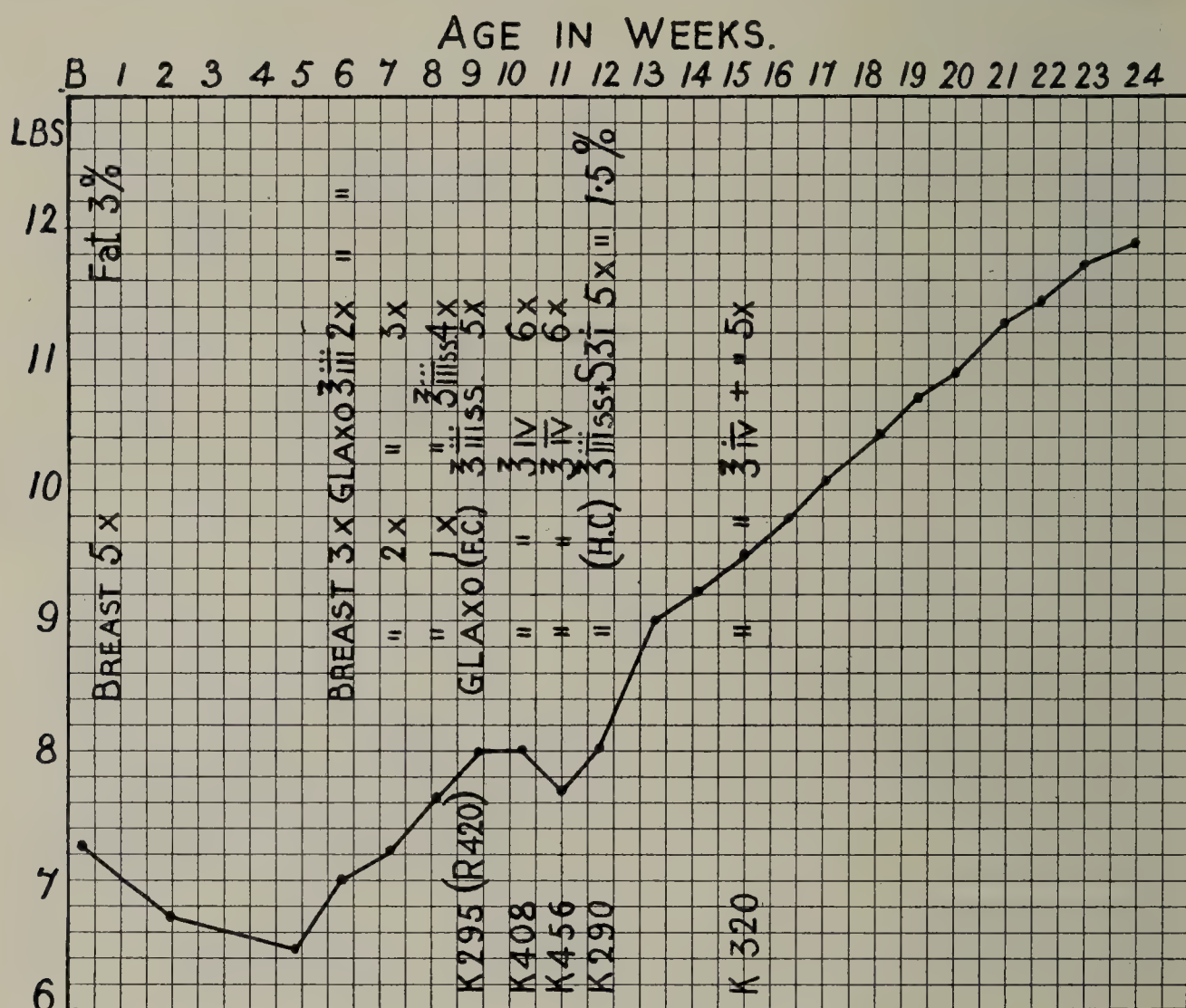


FIG. 68.—Case of Fat Dyspepsia, showing regular increase in weight on diminishing fat-intake. The child was healthy at birth and the mother seemed to have plenty of milk. Infant cried after each feed and brought up a mouthful.

Glaxo (F.C.) = Full-cream, and Glaxo (H.C.) = Half-cream milk. K = calories supplied per diem, and R = calories required per diem.

dyspepsia appears, or those of excessive carbohydrate feeding (dyspepsia with fever and loose undigested stools). It is thus seen how one condition reacts on another, and how intermixed are the different varieties of nutritional disease. There is produced a veritable vicious circle.

The parenteral infection may be *tuberculosis*, some *chronic suppurative disease of the ear or of the urinary tract*. Hence the need for a most thorough physical examination. The French

consider that *syphilis* is a common cause, but in our experience¹ lues, unless in an overt form, is very seldom responsible for nutritional disease.

In the varieties of marasmus due to the above causes the *treatment* is that of the primary mischief, with, in addition, a reduction in the diet so far as the fat and carbohydrate are concerned, or the presentation of a milk mixture favourable to the optimal action of the gut. Such a type of diet is to be found in *protein* or *albumin milk*.

Children seldom die of marasmus *per se*. In the most severe grades sudden death from no apparent cause may occur, but as a rule the fatal termination is due to some intercurrent infection, *e.g.*, pneumonia, pyelonephritis or enteritis, to all of which, as we have already remarked, the child has been rendered specially susceptible and little resistant. The particular infective condition may in consequence of the appropriate treatment be recovered from, but the child is usually left with a still more reduced tolerance to food and resistance to disease. The dieting in such cases is often extremely difficult, and it may be that breast-milk alone can save the child. If this is not available then protein milk is the best substitute, but care must be taken not to curtail the amount of food too much, as marantic children bear starvation badly. This must be specially borne in mind in the treatment of enteritis superimposed on marasmus. The period of starvation must in these circumstances be omitted and food and fluid must be given from the moment the child comes under observation. If sufficient cannot be given by the mouth, glucose intravenously and guarded by insulin (one unit of insulin for every two grammes of sugar) must be administered.

The special susceptibility of these marantic infants to infection and their impaired circulation demand careful nursing. Care must be taken that the children are not left lying in their cots too long, seldom being changed, and never nursed. Nursing and mothering are most important assets in the therapeutic regime. It is the difficulty of supplying these facilities, as well as the segregation of so much susceptible material and thus fostering the spread of infection, which makes the institutional treatment of this type of disease so unsatisfactory.

¹ L. Findlay, *Amer. Journ. Dis. Child.*, 1924, xxviii., 133.

GASTRO-ENTERITIS, DIARRHŒA, CHOLERA INFANTUM,
AUTO-INTOXICATION AND INTOXICATION

In this type of nutritional disease there are always evidences of gastro-intestinal irritation and fever, with, in the severer forms, apparent manifestations of toxic action. The mildest form Finkelstein would designate *dyspepsie*, when the illness is characterised by vomiting, undigested and frequent stools, and slight fever, with or without loss of weight. The child, however, is not gravely ill; there are no nervous phenomena; he is quite conscious and interested in his surroundings.

In the severer forms no better term than *intoxication* describes the picture. The child truly is as if poisoned. He lies in bed more or less unconscious, staring as if into space, every now and then emitting a sudden cry (hydrocephalic); the face is grey with perhaps some cyanosis of the nose and ears; the eyes are sunken with the corneæ glazed, and the nose is pinched (Hippocratic facies); he is restless, with occasional muscular twitches, and the breathing is deep and rapid and accompanied at times by action of the *alæ nasi*, so that some pulmonary mischief is suspected. There is usually fever, in fact there may be hyperpyrexia, and the pulse is small and rapid. Owing to the loss of fluid from vomiting and diarrhœa there is great loss of weight. In no other condition does there occur such a rapid loss of weight— $\frac{1}{2}$ or even 1 lb. in the course of twenty-four hours. It is this loss of fluid which is responsible for the sunken eyes and pinched nose (Hippocratic facies) and for the depressed fontanelle. The subcutaneous tissues become depleted, so that the skin hangs in folds on the limbs and abdomen and is devoid of elasticity, and when pinched up remains as a fold unduly. At times, and especially in the grave cases, a condition of *sclerema* develops, in which the skin and subcutaneous tissues feel as if made of rubber and adherent to the underlying bones. The skin cannot be pinched up between the finger and thumb; there is no pitting on pressure. This condition of *sclerema* is of very grave prognostic omen.

The tongue is dry and furred. The child may evince great thirst and is always eager to drink, but if the amount of fluid permitted at one time is not small it is almost at once rejected.

The excretion of urine is diminished, and acetonuria and albuminuria are not uncommon. Renal casts are often present

in the urine, and at times are so abundant as to form a deposit suggesting pus to the naked eye. Glycosuria may also be met with.

The blood reveals a moderate leucocytosis. There may also be a lowered CO_2 content confirming the presence of acidosis, which is suggested by the deep and rapid breathing (air hunger) and the presence of acetone in the urine (see pp. 556 and 564).

Between these two extremes all degrees of the process may be met with, and the milder varieties, if not immediately treated, are prone to pass into the more severe.

Causes of Dyspepsia and Intoxication.—Diet is undoubtedly a factor in the production of this condition. Over-feeding, and especially over-feeding with carbohydrate, leads to fermentation in the gut with irritation of the mucosa and diarrhœa. From this cause, however, seldom does any grave illness arise, but the child in consequence has been rendered more susceptible to infection, and, with the onset of a complicating otitis or pyelonephritis, a severe and dangerous attack may be precipitated. And since as a result of the damage to the intestinal mucosa the child's tolerance to diet has been reduced, any further dietary error is attended with the greatest danger.

Contaminated food, or at least food which is not strictly fresh, and which has become infected from much handling and being left exposed to the air, is also a cause not only of the mild but of the severe forms of the malady. It is truly remarkable how quickly a young infant may develop a severe diarrhœa and appear on the verge of death after a feed of milk which has "turned." Indeed, it is because of the difficulty of keeping the milk clean that diarrhœal diseases, at least the severer forms, are almost entirely limited to bottle-fed children, and, as these difficulties are greater during warm weather, that diarrhœal diseases are most frequently met with during the summer. So striking is this latter fact that a commonly employed synonym for the condition is "summer diarrhœa."

That the milk had become contaminated in some way has generally been considered the cause of the severe epidemics of *summer diarrhœa*, *cholera infantum*, or *British cholera*. Until some eight or ten years ago this disease was the greatest menace during infancy. In recent years, however, it has in great part disappeared and certainly has not appeared in epidemic form. In fact, it is the disappearance of epidemics of gastro-enteritis which is chiefly responsible for the fall of the

infantile death-rate in England during recent years, since it more than any other cause of death influenced the trend of the infantile mortality rate curve.

The particular error in the diet or nature of the contamination which induced epidemic diarrhoea has, however, never been discovered. The fact that when epidemics did occur they did so more or less all over Europe, and were thus more of the nature of pandemics, raises the question of some specific infective agent. Nevertheless, the inability to find any particular parasitic factor still leaves the matter in doubt. Indeed, if it be due to a specific organism, it is remarkable that breast-fed children should be so seldom attacked. Although their diet is obtained direct from the source and almost invariably sterile, the frequency with which organisms must be introduced into the mouth of the breast-fed child by its own hands, or by those of its nurse or playmates, and along with any article that it can lay its hands upon, makes it difficult to understand how he so frequently escapes. It may be, of course, that it is a question of mass dosage, and that the particular parasite requires a special medium, *e.g.*, cow's milk, for its development.

Attempts have been made to correlate the epidemics of gastro-enteritis with the incidence of flies, which are without doubt great disseminators of disease. Although this has not been completely successful it is worthy of note that in recent years in Britain there has been a simultaneous decrease in the number of flies and in the occurrence of this disease in epidemic form.

The fact that the disease in epidemic form was more severe in warm climates than in cold climates, and more prevalent in warm dry summers than in cold wet summers, undoubtedly points to temperature being in some way an influencing factor. It may be simply that during warm weather food is more likely to decompose, and that there is an increase in the number of flies which will disseminate any infection present. If, however, this were the explanation then the acme of the death-rate should follow some time after the acme of the temperature, because after the food had decomposed it would take some time for the disease to develop. The German school, headed by Finkelstein¹ and Rietschel,² pointed out

¹ H. Finkelstein, *Deut. med. Wochens.*, 1909, p. 1377.

² H. Rietschel, *Zeitsch. f. Balneo. Klimat. und Kur. Hyd.*, 1910, iii., No. 7.

that this was not so, and that the acme of the death-rate coincided with the acme of the temperature. Hence the suggestion that the temperature itself was perhaps the exciting factor and that the condition was of the nature of "heat stroke." As Rietschel says, "the high surrounding temperature in the homes of the poor is the most important link in the chain of damaging influences for the infant during summer."

Some of the more mild examples of gastro-enteritis or intoxication are caused by some infective process, the seat of which is far removed from the gastro-enteric canal. Of these, otitis is considered by some authorities the most important. Pneumonia and pyelonephritis are also not infrequent causes. The fact, however, that these infections occur more frequently during the winter months is rather against them playing any serious part in the causation of gastro-enteritis, which, as noted above, is eminently a disease of the summer months of the year. The symptoms are no doubt in part caused by the toxins produced locally, but by damaging the intestinal mucosa the train of manifestations consequent on primary intestinal disease is superadded, so that it is impossible to differentiate from the clinical picture the one variety from the other. One can here once more only insist on the importance of a complete physical examination in order to discover if any local mischief requiring attention is present. By noting the relative time of appearance of the gastro-intestinal symptoms and of the toxic symptoms it is often possible to tell which are primary and which are secondary. If the diarrhoea appears after the fever and the toxic symptoms, it is likely that some parenteral infection is the source of the mischief, whereas if the diarrhoea precedes or is synchronous with the fever the gastro-intestinal symptoms are probably primary. No stress can be laid on vomiting as this is such a frequent initial symptom of all disease during infancy.

One of the most striking features in the life history of this condition is that it seldom occurs in the absolutely normal child. Most frequently it is simply the terminal phase of a long-drawn-out illness, characterised by recurrent attacks of diarrhoea or dyspepsia from which the child has never completely recovered. He has always been left under-weight with a lowered resistance to infection and diminished tolerance for food—in short, marasmic—and with each attack of dyspepsia or intercurrent infection matters have further deteriorated. It

is this fact which makes one doubt if there is any specific infective agent at work and conclude that it is the result of the summation of many adverse factors. It is equally striking that it is a disease almost wholly of the poorer classes, in which of course adverse hereditary factors, adverse housing conditions, and deficient care all play their part. And, as previously mentioned, the disease is almost entirely limited to the bottle-fed child or the child receiving both breast and bottle. From these various considerations the important lessons to be learned are, that everything should be done to encourage breast-feeding, that milk for infant feeding should be of the purest and specially cared for, and that any digestive trouble is never too slight to receive the most careful attention.

Pathogenesis.—We have seen that the symptoms are of two distinct types, those the result of gastro-intestinal irritation (vomiting and diarrhœa), and those of a nervous nature (coma and twitchings). As a rule these two sets of phenomena are more or less commensurate, but they are not necessarily so. The marked toxic and nervous symptoms may occur in the absence of either vomiting or diarrhœa, and it is for this reason that they have been ascribed to the absorption of the products in the bowel (auto-intoxication). Nevertheless, the inability to demonstrate any toxic action of the intestinal contents by animal inoculation renders this explanation doubtful. It was suggested by Mellanby that the toxic factor might be of the nature of an amine base, and Brown¹ believes that he has experimental evidence of such being produced in the mucous membrane of the bowel itself.

The high fever, the diminished protein retention, the albuminuria with casts, and the leucocytosis would suggest that some infective process is at play. The tendency in hospital for groups of cases to occur either spontaneously, or soon after the admission of a similar case, supports such a contention. But the failure to find any specific bacterial flora is certainly an objection to the adoption of this theory of its cause. However, when we appreciate that many cases result from infection far removed from the bowel, *e.g.*, otitis media, the absence of positive toxic or bacterial findings in the gut is readily understood.

We have seen that in many cases the breathing is rapid and

¹ A. Brown and G. Boyd, *Canad. Med. Assoc. Journ.*, 1923, xiii., 800.

deep, and that there is acetone in the urine with a definite fall in the CO_2 content of the blood, and at one time it was the fashion to put the toxic symptoms down to acidosis. That there is a tendency to acidosis, as in almost all diseased conditions, there is no doubt, but it is exceedingly questionable if this plays the major part in the production of the clinical picture. The fever and the starvation, which are essential features of the illness, would in themselves cause a mal-combustion of fat and excessive production of acetone bodies. But the degree of acidosis is often no more marked than in many slighter ailments and the administration of alkalies (*per os* or intravenously), while counteracting the acidosis, has little effect on the toxic manifestations of gastro-enteritis.

Although vomiting and diarrhœa are not necessarily present, the most severe examples are usually accompanied by loss of fluid in this manner. Some writers believe that all the symptoms can be explained on this basis; hence the introduction of the term *anhydræmia*. In the examples unaccompanied by vomiting and diarrhœa, *e.g.*, those secondary to otitis media, influenza, and pneumonia, it is suggested that the permeability of the gastro-intestinal capillaries has been so altered that absorption has ceased and there has been a great outpouring of fluid into the intestine. In our experience the examples due to otitis media or other parenteral infection are not accompanied by severe anhydræmia. This indeed forms an important differentiating feature between the enteral and parenteral varieties of the disease. Undoubtedly many of the symptoms, *e.g.*, the collapse, the emaciation, the loss of elasticity of the skin and subcutaneous tissues, and the impaired circulation, are, or at least can be, induced by loss of fluid, but one cannot forget that in marasmus unaccompanied by toxic symptoms there is often as great a loss of fluid. In these circumstances, however, the loss of fluid has been more gradual, and it is possible that in this way one can account for the difference. Time has been given for the metabolism to accommodate itself.

Treatment.—Since we are ignorant of the true exciting cause, our therapy must be symptomatic in nature. We are, however, aware of certain metabolic errors, and we can do our utmost to rectify these. The child is being starved of food, salts, and water, and our remedial measures consist in an attempt to make up for these deficiencies.

If one appreciates the various causes which give rise to the condition the requisite therapy is apparent. The first line of treatment is that of the removal, if possible, of the primary cause. Naso-pharyngitis, tonsillitis, otitis media, and pyelonephritis all require their appropriate treatment.

Concerning the question of operative interference of otitis there prevails much difference of opinion. Some authorities,¹ e.g., Marriott, believe in incision of the *membrana tympani* whenever there is evidence by bulging of the membrane of an accumulation of pus within the middle ear, and in opening of the mastoid cells if there is extension of the mischief in this direction as shown by redness or œdema over the mastoid process. On the other hand, a pædiatrician of such wide experience as Finkelstein² is rather against surgical interference. Our own experience supports that of Finkelstein.^{3, 4} Although we have practised myringotomy in numerous cases, and mastoidectomy in not a few, seldom have we seen any striking results. The thinness of the membrane in the upper and posterior quadrant (Shrapnell's membrane) in the infant, and the ease with which it ruptures, would theoretically speak against the necessity of any surgical intervention. Those who believe in myringotomy consider that the operation renders less likely a spread to the mastoid cells, which is probably the really serious danger of the condition. In our experience this only occurs in a small minority of the cases. Of fifteen consecutive post-mortem examinations on children in whom there was otitis media the mastoid cells were involved in only four.

The gastro-intestinal manifestations are to be treated by dietetic measures. Whatever the cause of the symptoms, whether the condition be one primarily of the alimentary canal, or secondary to some infective process, the child's tolerance to food is greatly reduced. This is most marked in the examples

¹ W. M'K. Marriott, *Infant Nutrition*, 1930, 273. M. Renaud, *Bullet et mem. société médicale des hôpitaux de Paris*, 1921, xlv., 1326, 1352, 1384. A. M. Alden and H. W. Lyman, *Trans. Amer. Laryng. Rhin. and Otol. Soc.*, 1925, xxxi., 67. H. W. Lyman, *ibid.*, 1927, xxxiii., 354. T. H. Odenal, *Arch. Otolaryngology*, 1928, vii., 623.

² H. Fingelstein, *Lehrbuch der Sauglingskrankheiten*, Berlin, 1905. Bd. ii., 603.

³ L. Findlay, *Arch. Dis. Child*, 1932, vii., 307.

⁴ D. E. S. Wishart, *Journ. Amer. Med. Assoc.*, 1930, xcv., 1084.

of enteral infection. In these latter abstention from all food for twelve or twenty-four hours should be first carried out. With this alone, and allowing nothing but water, which should be given *ad libitum*, the toxic symptoms in all but the most severe cases will usually disappear. Vomiting may interfere with the water being retained, but if it is given in small quantities a certain measure of success will frequently be attained. A poultice applied to the epigastrium at times seems to help in controlling the vomiting.

Complete starvation is not so essential in the examples of parenteral infection, but it is nevertheless wise to reduce the amount of food, and any that is given should be of the blandest nature. Starvation, as already remarked, must be carefully carried out in those cases of intoxication superimposed on severe marasmus.

As dehydration is at least a prominent feature of the cases, whether it be the cause of the symptoms or not, it is imperative that the loss of fluid be replaced. This, of course, is attempted by giving the child water to drink *ad libitum*, but as a rule the amount ingested in this way is limited. Vomiting often defeats our object and also absorption from the gut is interfered with. In these circumstances the fluid must be given subcutaneously, intraperitoneally, or intravenously. It has been mentioned that an important feature of the illness is deprivation, or even a loss of salts, and hence saline solutions of isotonic strength should be employed. Normal saline (.9 per cent. NaCl) or Ringer's solution (NaCl .7 per cent., KCl .01 per cent., and CaCl_2 .02 per cent.) are both suitable solutions. Hypertonic saline solutions (NaCl 1.7 per cent.), as recommended many years ago in Asiatic cholera, with the idea that there will result a better retention of fluid, have been tried, but in our experience are not satisfactory. With hypertonic solutions we found an increased tendency to the development of pyelonephritis, which is an ever-present danger in this condition. This increased tendency to infection of the urinary tract we ascribed to the very defective flushing of the urinary tract in consequence of the diminished excretion of urine.

100 to 200 c.c. of one of the above recommended solutions can be administered subcutaneously into the abdominal wall or loose areolar tissue of the axilla. The serious disadvantage of the subcutaneous route is that it is painful, and tenderness

and induration of the parts remain for some considerable time afterwards, and in consequence seriously interfere with any physical examination. For these reasons, and also because larger amounts can be given, the intraperitoneal route is the one of choice. By this method as much as 500 c.c. can be given to a small baby. The operation can be carried out in either of two ways. An ordinary exploring needle, or one such as is used for the injection of serum, is the most convenient size. To this is attached a funnel by means of a piece of rubber tubing and the whole filled with the solution to be used, at the temperature of the blood. The needle may be inserted directly through the abdominal wall at a point midway between the *umbilicus* and the *symphysis pubis* in the middle line, care being taken to empty the bladder beforehand, for if this viscus is distended it will come within the field of operation and may be injured. Another technique may be employed. The abdominal wall is caught up between the fingers and thumb of one hand, and when one is certain that no coils of intestine are included it is completely transfixed with the needle, the fold is then released and the distal portion of the needle slips back into the cavity. The fluid is then slowly introduced under slight pressure.

Fluid may also be introduced by the intravenous route. The longitudinal sinus which is easily entered through the anterior fontanelle is the seat of election for the injection. The needle, the point of which should be ground at an angle of 45° , already attached to the funnel and filled with the fluid so as to avoid the introduction of air, is introduced at the posterior angle of the fontanelle in the middle line at an angle of 30° pointing towards the occiput. For this operation a two-way syringe (Lauer-Kaufmann) will be found of great help. When the syringe fills with blood one knows that he is properly within the sinus. The fluid is allowed to flow at a low pressure. By this means, however, only relatively small quantities of fluid can be introduced, never more than 20 to 30 c.c. per kilo body weight. This route has the further disadvantage that fluid introduced in this way is excreted very quickly.

By the introduction of fluid the dehydration disappears, the colour improves, the breathing becomes easier, the pulse stronger, and there is an increase in weight, so that often within a matter of a few hours a child apparently moribund looks comparatively well.

Starvation in this disease is not practised because starvation *per se* is curative. On general principles further starvation is bad, for the child has already been living on his own tissues, but it is practised simply because the intestine is not in a state to digest and absorb food. The damaged intestine requires rest. Hence to administer food by another route is advantageous. This is frequently done by the administration of a solution of glucose intravenously, the longitudinal sinus again being the vein of election. A 5, 10, or 20 per cent. solution of glucose can be used but, as mentioned above, in amounts not exceeding 30 c.c. per kilo body weight. Unfortunately, much of the glucose introduced in this way is lost by being excreted in the urine, but this can be avoided if one unit of insulin for every two grammes of glucose be injected at the same time. The injection of the glucose, just as that of saline, can be repeated not only during the period of starvation but also for some time thereafter. This is a matter which entirely turns on the state of the child and rate of recovery.

Blood transfusion has been recommended, but in our experience has not given very encouraging results.

After the period of twenty-four hours' starvation, food may be given by the mouth. Breast-milk is the ideal diet in this condition, but unfortunately, at least in this country, it is seldom available. If, however, breast-milk can be obtained, it is to be given in the quantities and at the intervals stated below. Next to breast-milk, protein milk is the food of choice. On account of its high protein content and relative poverty in salts and sugar, it counteracts fermentation and permits of the intestinal mucosa functioning at its optimum. After the period of starvation this milk should be given for the first day in half-ounce quantities every four hours, and then gradually increased by the addition of an extra half-ounce per feed every day. When the bulk of the feed proportionate to the child's age has been reached then sugar can be added, *e.g.* \mathfrak{Z} ss per feed, and later \mathfrak{Z} i and even \mathfrak{Z} ii per feed. When all symptoms have disappeared and the motions are normal the usual food, *e.g.*, undiluted milk, can be given. Instead of gradually increasing the nutritive value of the protein milk by the addition of sugar, a very satisfactory method is to replace the protein milk with undiluted cow's milk at the rate of half an ounce per feed per day. Thus, if the child were receiving \mathfrak{Z} vi of protein milk per

feed, he would receive $\frac{3}{4}$ ss protein milk and $\frac{3}{8}$ ss of undiluted cow's milk, then $\frac{3}{4}$ v protein milk and $\frac{3}{8}$ i of undiluted cow's milk per feed, and so on until he is getting only undiluted cow's milk. This method has the advantage of gradually acclimatising the child to a normal diet. The whole process may take a week or ten days, the rate of advance being governed by the disappearance of toxic manifestations and the behaviour of the motions. At first there may be no increase in weight, which is not surprising in view of the small amount of milk the child is receiving. Increase in weight may not result till he is being given a normal amount of food. Nevertheless, because so much of the loss of weight is due to loss of fluid, there may be, even with the smaller feeds, a definite gain in weight.

It is contended that undiluted lactic-acid milk is as well borne by these children as protein milk. Such, however, has not been our experience, but in view of the opinion of many writers it may be tried. This variety of milk is administered in the same quantities and at the same intervals recommended for protein milk.

Drugs in this disease are seldom of much value. Purgatives are rarely necessary, for by the time the patient comes under observation the bowel is already well cleared out. If the case is seen very early castor-oil may be administered. Astringents to control the diarrhoea are seldom of much avail. If the diarrhoea is very profuse opium can be tried. Kaolin in our experience is of little value. Stimulants may be necessary, and in our experience brandy, in suitable doses (10 to 15 minims) well diluted, is as satisfactory as any. Alcohol has the advantage of being at the same time a food and a nerve sedative as well as a cardiac stimulant.

ILEO-COLITIS, DYSENTERY, INFECTIOUS DIARRHŒA

We have already mentioned that bacteriological investigations had enabled us to identify this disease as a clinical entity (p. 203). Ileo-colitis is characterised clinically by the passage of "dysenteric stools," pathologically by a catarrhal or necrotic and ulcerative state of the large bowel and lower end of small intestine, and bacteriologically by the presence of organisms of the dysentery group.

The disease occurs sporadically at all seasons of the year, although it is undoubtedly met with most frequently during the warmer months. The mensual incidence in 127 examples occurring in R.H.S.C., Glasgow, was as follows:—

Mensual Incidence of Ileo-colitis.

Month	No.	Month	No.
January	2	July	15
February	6	August	15
March	3	September	20
April	10	October	16
May	13	November	10
June	13	December	4

Epidemics, however, especially in Children’s Homes and Hospitals, have been recorded, and on occasion all the members of a family are attacked.

Boys and girls are almost equally affected. Of the above 127 cases, 74 were boys and 53 were girls.

Children of all ages are susceptible to the infection, but it is most frequently encountered during the first two years of life. The age incidence in the above 127 examples was as follows:—

Age Incidence of Ileo-colitis.

Under 1 year	38	Under 8 years	5
„ 2 years	42	„ 9 „	1
„ 3 „	16	„ 10 „	1
„ 4 „	14	„ 11 „
„ 5 „	6	„ 12 „
„ 6 „	1	„ 13 „
„ 7 „	2		

Koplik also found that the second year was that of greatest incidence.

Symptoms.—The illness may set in suddenly with fever in the midst of good health, or it may succeed an attack of chronic diarrhœa. So frequently does the latter happen that it would seem as if chronic enteritis renders the gut more easily attacked by the *B. dysenteriæ*. The diarrhœa even in the primary cases may in the early stages present nothing characteristic, but as a rule comparatively soon typical dysenteric stools make their

appearance. These are small, consist for the most part of blood and mucus, and are evacuated at frequent intervals with much straining. Vomiting may be present at the onset but it is not the rule.

Fever of variable degree (100° to 104° F.) is always present.

The illness reaches its height within twenty-four to forty-eight hours. The child is acutely ill, is severely toxic with nervous phenomena (coma or convulsions), highly fevered with a dry pungent skin and dry furred tongue, and perhaps a smell of acetone from the breath. The eyes are sunken, but there is as a rule no marked degree of anhydræmia which is so characteristic of gastro-enteritis. Toxic symptoms and nervous phenomena are almost invariably present. The severe nervous manifestations (coma and convulsions) are specially prominent in the well-nourished and obese child of the so-called thymolympathic nature. In some of these very toxic examples the disease runs a most acute course, death supervening within twenty-four or thirty-six hours and before any diarrhœa has appeared to give a clue to the nature of the malady.

The abdomen is slightly distended and tenderness may be elicited along the course of the ascending but more frequently over the descending colon. The motions are frequent, small, slimy, of a fetid odour, and composed almost entirely of mucus, blood and pus. Tenesmus of varying degree accompanies the bowel movements. Microscopic examination of the stools reveals the presence of red blood corpuscles, pus cells, and microphages. During the first days of the illness phagocytosis of Gram-negative bacilli is observed, and bacteriological examination reveals the presence of organisms of the dysentery group—chiefly of the Flexner type. In the early stages these organisms are easily isolated from the motions, but later on they are difficult to discover and atypical dysentery organisms (*B. Morgan*, *B. fæcalis*, *B. coli ærogenes* and *paracolon* types) make their appearance. It is probably because many cases have been investigated only late in the disease, when these latter types of bacilli have been isolated, that there exists so much doubt regarding the specificity of ileo-colitis during infancy and childhood.

The urine generally contains acetone and a haze of albumin, and rarely blood and pus.

A moderate leucocytosis may be present, but is by no means

invariable. Although specific agglutinins are developed this rarely occurs before the twelfth day, and as even normal sera may agglutinate *B. dysenteriæ* (Flexner) in dilution of 1 in 150 this reaction is of little value in diagnosis.

When reacting to treatment the disease usually runs a course of ten to twelve days before the motions become normal. The blood disappears first, while the mucus and pus persist. Complications such as broncho-pneumonia and pyelonephritis are liable to occur and these help to swell the death-rate, though a fatal issue may ensue from the toxic effects of the disease *per se*. During infancy the death-rate is very high, but it falls considerably during the second year, and is comparatively low during the later years of childhood. In the 127 cases occurring in R.H.S.C., Glasgow, the death-rate was as shown below :—

Death-rate in Ileo-colitis.

Age.	Death-rate. Per cent.	Number of Cases.
Under 1 year. . . .	65·7	38
1 to 2 years	23·8	42
3 „ 5 „	22·2	36
6 „ 13 „	9·0	11

The **diagnosis** rests on the presence of the dysenteric stool, which is small, consists of mucus, blood, and pus, and is evacuated with much straining. If the case is seen late in the course of the disease blood may be absent, as this is the first to disappear, but pus will be present. For the detection of the pus microscopic examination is useful. Bacteriological examination of the stool in the early stages will reveal the presence of an organism of the dysentery group—most frequently of the Flexner type. Cruickshank¹ considers the presence of a dysentery bacillus typical of ileo-colitis, and most English workers have found it seldom in any other type of enteritis.² In America,³ on the other hand, dysentery bacilli are reported frequently in

¹ J. Cruickshank, *Quart. Journ. Med.*, 1925, xviii., 339.
² D. Nabarro, *Brit. Med. Journ.*, 1923, ii., 857.
³ Flexner and Holt, *Rockefeller Inst. Monograph on Epidemic Enteritis*, 1904.

ordinary enteritis and even in normal stools. Cruickshank, working in Glasgow, did not find this organism in non-dysenteric diarrhoeal or normal motions.

The condition may be mistaken for intussusception, but the relatively severe toxic symptoms, the absence of colic and of an abdominal tumour, usually enable the correct diagnosis to be arrived at. In doubtful cases it is wise to explore the abdomen and rectum thoroughly under general anæsthesia.

Treatment.—In the early stage of the disease it is wise to administer a dose of castor-oil in order to clear out the bowel and to institute a period of starvation for a matter of twelve hours, during which time water alone is given. At the end of this time boiled milk, lactic-acid milk, or best of all, protein milk may be allowed. It is not necessary to restrict the amount of food in this condition as is done in cholera infantum, because the great extent of the bowel is unaffected. Feeds of 3 oz. may be commenced with in the youngest infant and more or less normal quantities in the older children. Magnesium sulphate should be given in three successive doses of 10 to 30 gr. each at hourly intervals each morning. This treatment, which has been found of great value in dysentery in the adult, should be continued till the motions have become normal. It would seem to act by causing one or two satisfactory evacuations per day, and in consequence there is much less tenesmus. As previously mentioned, the blood disappears first. The motions may be normal within four to five days and as a rule within ten days.

Astringents and opiates *per os* are to be avoided, as also lavage of the bowel and injections of tannic acid, at one time such a favourite procedure in this disease. Since the introduction of the treatment with magnesium sulphate we have found lavage of the bowel unnecessary.

If the child is very toxic, anti-dysenteric serum should be administered. On occasion we have seen this do much good, but of course it is only efficacious when the antiserum is specific against the particular organism causing the disease. In order that there may be a greater chance of this happening polyvalent sera are employed.

It has already been remarked that the dysenteric patient is seldom dehydrated, as in the case of gastro-enteritis, and hence subcutaneous or intravenous injections of saline are as a rule

not called for. Should, however, there be great loss of fluid, and especially if there is much vomiting, saline solutions intra-peritoneally and glucose solutions intravenously must be resorted to.

It is important to remember that as this disease is infectious the child should be isolated and all excretions be thoroughly disinfected before being disposed of.

CHAPTER X

INFANTILE SCURVY

(*Barlow's Disease*)

INFANTILE scurvy is a state of perverted nutrition which affects all the tissues and organs more or less, arresting growth and leading to anæmia, local œdema, and hæmorrhages—especially under the periosteum—and to disturbance of most of the systems and functions of the body. Its presence also gives rise to a definite lessening of the resistance of the tissues to various forms of infection. It is specially worthy of study because it is easily overlooked in its early stages, entirely preventable, very amenable to simple treatment, and, when left untreated, most distressing in its symptoms, and often dangerous to life.

Glisson, in his classical *Treatise on the Rickets*,¹ gave an excellent description of the symptoms of infantile scurvy, recognised their nature, and showed that they were not rachitic in origin but signs of a scorbutic complication. His work on the subject seems, however, to have been entirely overlooked for more than two hundred years, for most of the papers describing the morbid anatomy of the disease, which appeared during the nineteenth century, ignored its scorbutic origin (Möller, Hirschsprung, T. Smith, Gee, and others). Ingerslev, in 1871,² and W. B. Cheadle, in 1878,³ pointed out the real cause; but it was only in 1883 that the symptoms and etiology of the condition were realised and accepted by the profession at large, as the result of a thorough and convincing investigation on the subject by Sir Thomas Barlow.⁴ During recent years much interesting and valuable work has been published on

¹ English translation of 2nd edit., 1651, 249. See Clin. Lect. on "Infantile Scurvy," by G. F. Still, *Brit. Med. Journ.*, 28th July 1906, 186.

² *Hospitalstidende*, 1871, 121, and *Virchow's Jahresber.*, 1873, 697.

³ *Lancet*, 16th November 1878, 685; *ibid.*, 15th July 1882, 48.

⁴ T. Barlow, *Trans. Med. Chir. Soc. Lond.*, 1883, lxvi., 159.

the subject in America—especially by A. F. Hess¹ and his collaborators, and in this country by Miss Chick and those who have worked with her.

Clinical Features.—Infantile scurvy is seen almost always in children between six and fourteen months, but it may occur in the third year or later, and also, though only very rarely, and in a mild form, as early as the fourth or fifth month. In the vast majority of cases the children have been taking a scorbutic diet for four or five months before the characteristic symptoms began.

It is far oftener seen among the upper and artisan classes, who are given to the continuous use of proprietary infant foods or to a strict routine of feeding, than among the very poor, who cannot afford them, or who vary their use by giving fragments of potato, green vegetables, fruit, and meat which, however indigestible, are antiscorbutic. For similar reasons it is mainly a disease of large towns, and is found chiefly where artificial feeding is frequent and fresh milk difficult to obtain.

It is seldom or never seen in breast-fed children. The great majority of such records do not bear scrutiny and the condition was probably sepsis or congenital syphilis. As Finkelstein says, there has been “no necropsy of a breast-fed case or conclusive X-ray picture.”²

Symptoms.—The most characteristic symptoms of infantile scurvy, being due to hæmorrhage, generally come on suddenly. They do not, however, set in in the midst of perfect health. Their appearance is always preceded for weeks by a slowly developing state of cachexia, which is characterised by increasing listlessness and debility with a rapid pulse, and disinclination for movement of any kind—the so-called *latent stage*. The child is anæmic, and in severe cases has a peculiar sallow, earthy tint; he is short of breath on exertion, cries readily, refuses his bottle, and often has slimy green stools with occasional traces of blood in them. At this time also there may be tenderness on movement or pressure, especially of the feet and legs—much more than would be accounted for by the rickets which is not infrequently also present.

If no change is made in the child's diet and surroundings,

¹ A. F. Hess, *Scurvy, Past and Present*, Phil., London, 1920.

² H. Finkelstein, *Lehrbuch der Säuglingskrankheiten*, Berlin, 3rd edit., 1924, 365.

pseudo-paralysis of one of the lower limbs sooner or later develops, more or less suddenly. The infant lies in bed with an expression of apparent terror, ceases to move the affected limb, and screams when it is touched, from fear that it is going to be moved, and sometimes apparently owing to actual local pain. When the femur is the bone affected, as most frequently happens, he lies on his back with the limb abducted and rotated outwards and the knee slightly flexed (Fig. 69). The affected part is much swollen and extremely tender; the skin is tense and shiny, and there is often a condition of hard

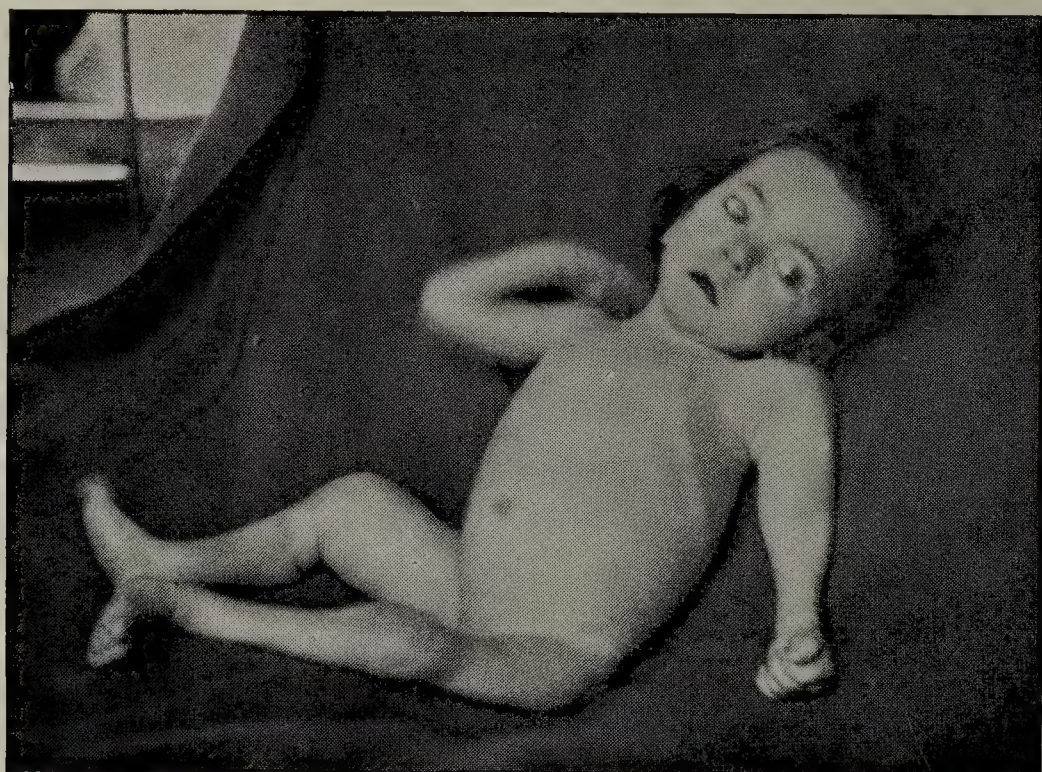


FIG. 69.—Infantile Scurvy. Subperiosteal hæmorrhage of right femur and left humerus.

œdema. In addition to the *periosteal hæmorrhage* which causes the pseudo-paralysis, effusion of blood and œdema may occur in the muscles and intramuscular spaces, and occasionally these tissues only are affected. There is no rise of temperature, and the joints are scarcely ever affected. In severe cases soft crepitus may be felt from *separation of the epiphysis*. Sometimes more or less spontaneous *fractures* of the shaft of the bone are the chief features of the disease (Figs. 70 and 71).

In many cases the thorax presents a remarkable and characteristic deformity, to which Barlow¹ first drew attention.

¹ See Articles in Keating's *Cyclopædia of the Dis. of Child.*, 1889, ii., 272 ; and in Grancher and Comby's *Traité des Maladies de l'Enfance*, 2^{me} edit., 1904, t. i., 909.

This consists in a depression of the sternum and costal cartilages, as if they had been pushed back, and it gives rise to a row of projections along each line of costochondral junctions, which is apt to be mistaken for a rickety rosary. This kind of beading, however, is not due, like that in rickets, to enlarge-



FIG. 70.—Skiagram of left fore-arm in boy of $1\frac{3}{2}$ years, suffering from Scurvy, showing fracture of radius and ulna. (From same case as Fig. 71.)

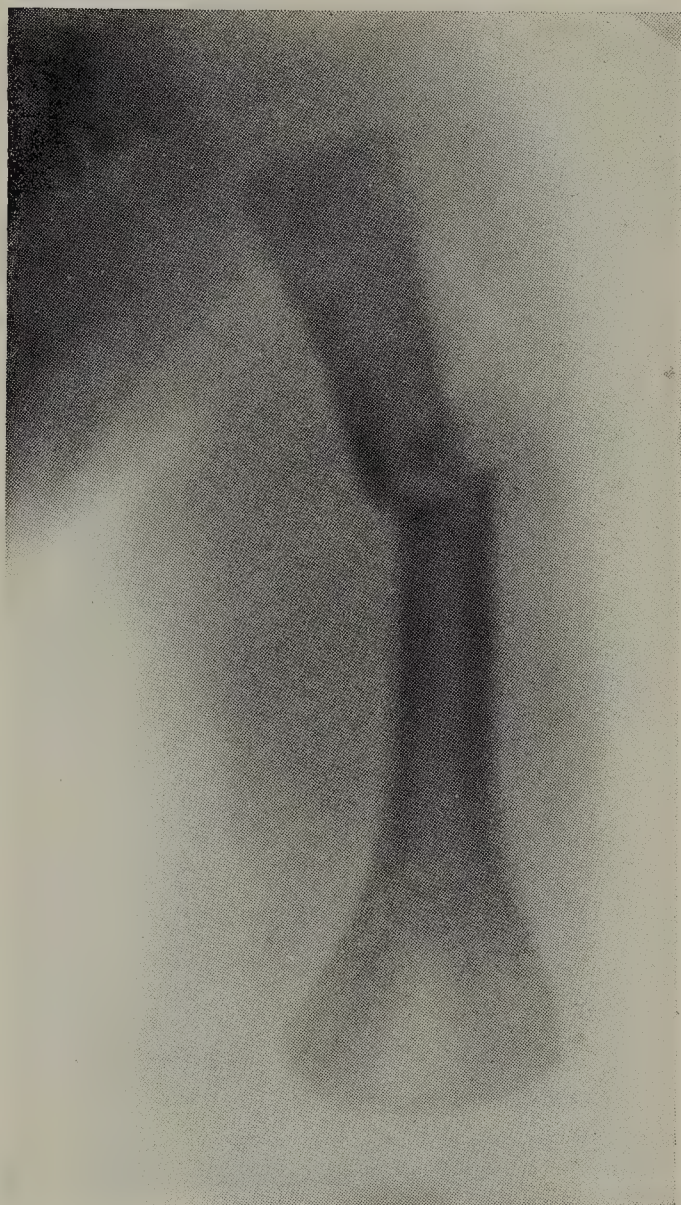


FIG. 71.—Skiagram of left humerus in boy of $1\frac{3}{2}$ years, suffering from Scurvy, showing fracture. (From same case as Fig. 70.)

ment of the ends of the cartilages, but to their dislocation forwards in advance of the ends of the bones.¹ It is one of the earliest signs of the disease to appear, and it usually disappears rapidly under treatment.

Much less commonly, the subperiosteal hæmorrhage takes place in the upper limb, on the pelvis or scapula, or on one or more of the ribs. In rare instances it occurs under the

¹ Still, *Common Disorders and Diseases of Childhood*, 4th edit., 1924, 131.

dura mater¹ and it takes place more frequently inside the orbit, in which situation it may cause considerable *proptosis* (Fig 75). The affection of the limbs tends to be symmetrical, that of one side following shortly after that of the other.

Changes in the bones can usually be appreciated by means of X-rays. Probably the earliest and most characteristic feature is the presence of the white line of Frænkel. This is a thin

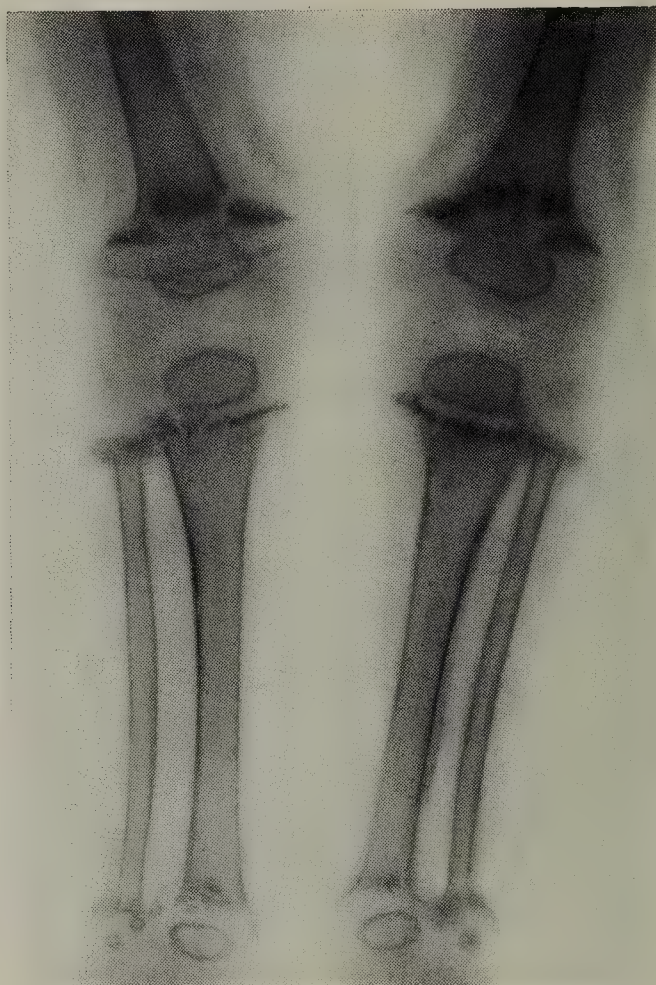


FIG. 72.—Skiagram of both lower limbs in case of Scurvy (boy aged 1½ years). Note presence of rickets (cupping and deficient calcification of epiphyseal ends of the bones), Frænkel's white line of increased condensation at epiphyseal end of the bone and extending beyond margin as a tag, subperiosteal hæmorrhage of left tibia, and "outlining" of the bones generally but especially of the epiphyses.

layer of increased density along the epiphyseal margin of the diaphysis and often present as a tag (Fig. 72) beyond the edge of the bone. When there is apparent effusion this tag is continuous with the outer limit of shadow and suggests that at this point there has been separation of the periosteum. Contrary to what might have been expected, the most marked shadows from effusion are seen late in the course of the disease, or even when clinically it has apparently recovered. This, of course, is due to the condensation, organisation, and calcification

¹ G. A. Sutherland, *Brain*, lxx., Spring 1894, 27.

of the effused blood (Figs. 73 and 74). The epiphyses are often unduly sharply defined and almost appear as if they had been artificially outlined. At times the only radiological evidence of the disease are multiple fractures (Figs. 70 and 71). Rachitic changes are not infrequently present at the same time (Fig. 72).

Along with the subperiosteal hæmorrhages, and often before them, cutaneous *ecchymoses* may occur. These have often the

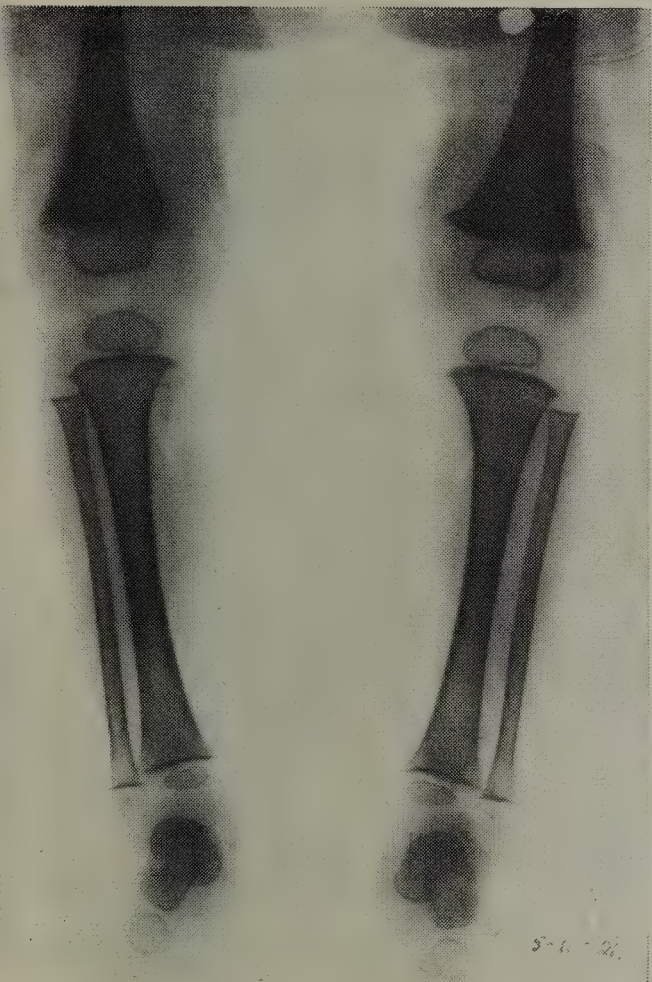


FIG. 73.—Skiagram of lower limbs in Scurvy (girl aged 8 months). Note Frænkel's line at epiphyseal end of diaphysis and the "outlining" of epiphyses. Child acutely ill at this time. (From same case as Fig. 74.)



FIG. 74.—Skiagram (in same case as Fig. 73) taken sixteen days later. Note the appearance of extensive subperiosteal hæmorrhage at lower end of right femur. At this time all pain and tenderness and much of the swelling of leg had disappeared.

appearance of ordinary bruises, and they are very easily produced by slight causes; sometimes they take the form of scattered petechiæ. The vaccination scar is a not uncommon site of such hæmorrhages. Another situation in which they are early met with is over the margins of the orbit, where they are probably due to too vigorous drying of the child's face after washing. As the mother uses her right hand in this process, the child's left orbit is that usually affected. Sometimes also

ecchymoses and œdema are present in the conjunctivæ, and eyelids and the deeper orbital tissues may be similarly affected (Fig. 75). Rarely *hæmatemesis* occurs, occasionally *epistaxis*, sometimes bleeding from the bowel, and very often *hæmaturia*.



FIG. 75.—Infantile Scurvy in girl of 12 months. Showing proptosis, and ecchymosis of the orbits.

It should be remembered that one or other of these hæmorrhages may be the only manifestation. Scurvy should always be considered in the case of any unexplained bleeding. In one child of eleven months, who looked the picture of health,

and who jumped about on his mother's knee, the only manifestation was a spongy and swollen condition of the gum, almost completely embedding the lower central incisors, and which had been mistaken for sarcoma.

As Hess and Fish have shown,¹ the hæmorrhagic tendency in scurvy is not directly due to the state of the blood, which is merely one of simple secondary anæmia with relative diminution of the hæmoglobin, but to a morbid change in the walls of the small blood vessels, which renders them liable to rupture from a moderate increase of pressure. It is for this reason that injury so frequently determines the so-called acute or hæmorrhagic phase of the disease.

In addition to the more or less hæmorrhagic symptoms, there are many indications of *minor hæmorrhages*, *œdema*, and *malnutrition*, in connection with the alimentary, circulatory, respiratory, urinary, and other systems of the body; and in many instances there are also symptoms of secondary septic infections of various kinds.

The *gums* are, at first, pale and normal in appearance; but on close examination, small submucous hæmorrhages are sometimes found on them, or on the vault of the hard palate. Later, if several of the teeth have appeared, the gums become *red*, *swollen*, and *spongy*, and are often ulcerated, but when there are no teeth the gums retain their normal appearance. Although no change is visible in the infant's teeth at the time of the attack, it is probable that their structure is always more or less seriously, if not permanently, damaged by the disease from absorption of the cement membrane of the alveolar sockets.

In connection with the circulatory system—the heart is often found to be *dilated* or *hypertrophied*, and attacks of tachycardia are not infrequent. These symptoms, and the *polypnœa* and *dyspnœa* which are also sometimes seen, may be due to œdema or some other disturbance of the pneumogastric nerve.²

The *urine* may be scanty, and when this is so, it is found to increase again on antiscorbutic treatment. Occasionally it is bright red with blood which apparently comes from the kidney, as numerous blood-casts are often found; in most cases some

¹ *Amer. Journ. Dis. Child.*, Dec. 1914, viii., 386; see also G. Marshall Findlay, *Journ. of Path. and Bact.*, 1921, xxiv., 441.

² A. F. Hess, *Proc. Soc. Experim. Biol. and Med.*, 18th Oct. 1916, xiv., No. 1, 4.

red corpuscles are found on microscopical examination. Pus may also be present from a *Bacillus coli* infection of the urinary tract. Even when no blood or pus is present, *albuminuria* is not uncommon.

Hess¹ has observed increased knee-jerks, superficial tenderness of the skin, and œdema of the optic discs.

In long-standing cases the disease seriously interferes with the growth in *weight*, and the weight remains stationary for weeks or months, whatever the amount of the calories in the food, unless the child has been previously underfed; and on antiscorbutic treatment it rises again and may be above the normal for a time. Scurvy differs from ordinary malnutrition in that it also causes a definite retardation in the growth in *length*.² This is, however, only temporarily in abeyance, and is resumed whenever treatment is begun.

A *high temperature* is not common in infantile scurvy, and when present to any degree it is usually due to some inflammatory complication such as acute pyelo-nephritis. It may occur, however, in some cases in which no local cause can be discovered.

The *impairment of the resistance* is shown by the frequency with which boils, nasal catarrhs, and infections of the urinary tract are encountered. Coli-pyelitis is so common, relatively, in scorbutic cases that it is always well, when it is present in any case, to investigate the nature of the diet.

When scurvy occurs in children over two years old, the symptoms resemble those in adults; subperiosteal hæmorrhages are much less likely to take place, and a spongy condition of the gums is always a prominent symptom simply because teeth are usually present at this age.

Etiology and Pathogenesis.—The determining cause of the disease is a deficiency in the diet of an accessory food factor—the so-called water-soluble substance or vitamin C—and the degree of the deficiency determines whether the disease will be latent or active. This deprivation or deficiency must be continued for some time, probably for more than four months, although the premonitory or latent state may develop even after three months' deficiency.

There is no doubt that there is an individual susceptibility

¹ *Journ. Amer. Med. Assoc.*, 18th Sept. 1915, lxx., 1003.

² A. F. Hess, *Amer. Journ. Dis. Child.*, Aug. 1916, xii., 152.

to the disease; otherwise it is impossible to explain how all children fed on a similar diet do not react alike. Other factors which seem to have a bearing on the development of this disease are injury and infection. A fall frequently precipitates an attack, but this is no doubt just in so far as the trauma damages the weakened vessels and determines the hæmorrhage. An infective condition like influenza has been known to cause epidemics of scurvy.

The antiscorbutic vitamin is a component of all fresh foods and among these can be included milk. In different foods, however, it is present in very varying amounts; while oranges are particularly rich in the vitamin, apples and grapes, on the other hand, contain only very small quantities. Thus the quantity of the particular food ingested, apart from its nature, determines the amount of the vitamin presented. It is important to bear in mind that this vitamin, being a living substance, is unstable and can be destroyed. First of all it tends to become inactive as the food ceases to be fresh—the so-called ageing; this is particularly important in the case of milk, which apparently only contains small quantities. The vitamin is also affected by heat, by handling, and by aeration and oxidation. The reaction of the food also influences the life of the vitamin, *e.g.*, an acid reaction tends to hinder and an alkaline reaction favours its disappearance.

Fresh milk from healthy cows has a distinct though not a very powerful antiscorbutic property; but there is reason to believe that this may be interfered with in the case of cows that are wholly stall-fed.

Simple heating of the milk on one occasion does not appear to lessen it appreciably, for it has been abundantly proved by long experience in France, and also in this country, that fresh milk may be kept at or about the boiling-point in a Soxhlet's steriliser, or other similar apparatus, even for as long as forty minutes, without losing its antiscorbutic value to any harmful degree. It is to be noted that, in this method of preparation, there is comparatively little handling or shaking of the milk, and very little exposure of it to the air. Hess has demonstrated that children fed on milk kept at 145° F. for thirty minutes do not develop scurvy.

If the milk is heated more than once, however, its antiscorbutic activity is probably always much diminished. This is

especially likely to occur if some time has elapsed between the heating process and feeding of the baby—for, as already mentioned, the keeping of milk for a long time also tends to impair its vital properties. Since dairy-keepers have realised that the heating of milk enables it to be kept longer without turning sour, there has been a definite increase of scurvy in many places—partly because the milk already heated in the dairy has reached the home in a far from fresh condition, and partly because it has often been boiled or pasteurised a second time before being given to the baby (Heubner,¹ Neumann,² Sutherland³). In some dairies in this country there is a pernicious habit of sterilising what is left over of the day's supply to make it keep, and adding this next morning to the fresh milk.

It has been recently pointed out that *oxidation* has a powerful effect in diminishing the antiscorbutic value of milk and other foods. Hess and Unger⁴ have found that milk to which hydrogen peroxide has been added in the proportion of 4 c.c. to a litre, rapidly causes scurvy in guinea-pigs. An epidemic occurred in Edinburgh during 1908 and 1909 from the use of "Buddeised Milk." The process used in preparing this milk (so as to sterilise it as far as possible) included keeping it between 51° and 52° C. for at least three hours and adding to each litre of it about 15 c.c. of a 3 per cent. solution of peroxide of hydrogen.⁵

It has also been shown conclusively by Hess⁶ and Zilva⁷ that, although simple heating has no bad effect, a dangerous degree of deterioration of the milk may occur if it is treated in any way—with or without heating—which leads to its being freely *aerated*. Such aeration is obviously apt to take place when milk that is boiled in an open pan is much shaken or stirred or even merely agitated by its own bubbling.

Another interesting point in this connection is worth mentioning, as in some countries it has a very practical bearing. Hess and Unger, *loc. cit.*, in view of the well-known action of catalysis in increasing oxidative processes, and the frequency

¹ *Berl. klin. Wochenschr.*, 1903, No. 13, 285.

² *Deutsch. Med. Wochenschr.*, 1902, 245.

³ *Brit. Journ. Child. Dis.*, May 1906, 229.

⁴ *Proc. Soc. Experim. Biol. Med.*, 1921, xviii., 143.

⁵ R. Tanner Hewlett, *Lancet*, 27th Jan. 1906, i., 209.

⁶ *Brit. Med. Journ.*, 31st July 1920, ii., 154.

⁷ *Lancet*, 5th Mar. 1921, i., 478.

with which traces of copper are found in milk, performed some experiments on guinea-pigs. They found that those animals that were fed on milk which had been *heated in a copper vessel* for forty minutes developed rapidly fatal scurvy, while others that were given the same milk heated similarly, in a glass vessel, remained quite unaffected.

Fresh milk carefully *pasteurised* or sterilised by boiling in the home very seldom causes scurvy; but, when the process is carried out commercially, on a large scale, the milk may suffer from the greater amount of "handling" that it gets.

The *peptonising* of milk greatly lessens its antiscorbutic properties; but *buttermilk* of any kind retains them fully.¹

Dried milks, such as "Glaxo" and "Cow and Gate" Milk, which are prepared by rapid heating, rarely cause scurvy. Those in the preparation of which the "spray process" is used are much more likely to do so.²

All the *proprietary infant foods* predispose to scurvy, and the same may be said of the different brands of *condensed milk*.

Bearing in mind these various influencing factors, it will be readily understood how one and the same system of feeding may not always be followed by the same result. For example, the age of the milk, whether it be heated or not, and the quantity ingested are particularly important in determining the amount of the vitamin available, and yet these are two factors which seldom receive consideration. It is probably because in the final mixtures made from proprietary foods there is so little milk, much of their nourishment being due to added carbohydrate, that scurvy so frequently follows their use. It may also be that in the very rare examples of scurvy in breast-fed children the quantity consumed is the important factor, since it is very doubtful if breast-milk is ever deficient in the necessary vitamin. At any rate it is a remarkable fact that in countries, *e.g.*, Russia, where scurvy is endemic among the adult population, it is practically unknown in the infant.

Diagnosis.—It is easy to diagnose a well-marked case of infantile scurvy; but in slight, chronic, and atypical cases, especially when the patient is suffering at the same time from

¹ J. D. Comrie (*Edin. Med. Journ.*, April 1920, xxiv., 214) reports very striking improvement in adult cases of scurvy in Russia from the use of milk which had simply been allowed to turn sour.

² Jephcott and Bacharach, *Biochem. Journ.*, 1921, xv., No. 1, 129.

some other disease, the condition is rather apt to pass unrecognised.

The possibility of scurvy being present must *always* be kept in mind whenever a child has been confined for more than four months to a diet either devoid or containing an insufficiency of fresh elements. If it has contained plenty of fresh material and raw milk, scurvy may be entirely put out of the question, however suggestive of the disease some of the symptoms may be.

Whenever a child, between six and eighteen months, who has been on a possibly scorbutic diet, shows *tenderness on handling of the lower limbs*, especially of the feet, without any history of injury, this should always arouse a strong suspicion of the presence of scurvy. If blood corpuscles are found in the urine on microscopical examination, this may be held to settle the diagnosis; and the rapid and permanent improvement which always follows the administration of a potent anti-scorbutic (orange juice) puts the matter beyond doubt.

Among the morbid conditions which are liable to be mistaken for infantile scurvy are fracture, acute periosteal abscess of the femur, and angioneurotic œdema of the thigh. On the other hand, scurvy is at times mistaken for poliomyelitis, rheumatism, or congenital syphilis.

Treatment.—(A) *Prophylactic.*—The means of preventing infantile scurvy consist in avoiding the various harmful methods of treating the milk which have been described above. All kinds of proprietary infants' foods and condensed and dried milks should also be avoided if possible, and, if used, should be discontinued after a few weeks. If, however, it is considered desirable for safety's sake to give the child pasteurised or sterilised milk, all fear of scurvy developing can be dispelled by the regular addition to the diet of a small quantity of orange juice or other reliable antiscorbutic. Half a teaspoonful of orange juice three or four times weekly will be found sufficient.

(B) *Curative.*—The treatment of infantile scurvy consists in bringing the patient under the influence of the antiscorbutic vitamin as quickly as possible. The rate of recovery bears a very definite relationship to the amount of the vitamin presented. When a sufficiency is given there is no more striking therapeutic effect than the reaction of a scorbutic patient to antiscorbutic treatment. Within twenty-four or forty-eight hours a complete transformation in the condition is brought about. The child,

previously fretful, lying with staring eyes, evidently afraid of being touched, and screaming if handled, becomes contented, smiles, and allows himself to be nursed without crying. In the matter of a week all clinical manifestations have disappeared. Radiological evidence, however, persists for a long time—in fact, it is several months before the bones assume a normal appearance (Fig. 74).

The most efficient vehicle of the vitamin is orange juice, and the child should be given as much as its digestion will tolerate. At least one half ounce and if possible one ounce daily is a suitable amount. Hess¹ has administered orange juice intravenously, which may be practised if the disease is far advanced or a sufficiency of the orange juice cannot be tolerated by mouth. The juice is boiled “for about five minutes and rendered neutral or alkaline just previous to its injection by the addition of normal sodium hydroxide.” In one case treated in this fashion Hess and Unger noted improvement sixteen hours after an injection of 6 c.c. These authors employed doses varying between 6 c.c. and 35 c.c.

If orange juice is not available, tinned tomato or lemon juice may be employed. According to Chick and Rhodes,² the juice of raw swedes is an efficient antiscorbutic. The clean cut surface of the turnip is grated with an ordinary kitchen grater, the pulp folded in muslin, and the juice squeezed out with the fingers. Other fruits, *e.g.*, apples, grapes, and limes, are unreliable in their potency and should never be employed therapeutically.

When the acuteness of the mischief has subsided an attempt must be made to put the child on a rational diet, *e.g.*, fresh cow's milk which has been pasteurised or sterilised at home, but the extra antiscorbutic must be continued for months. Although clinically a cure may soon be apparent, months are required to allow the bones and viscera to return to normal.

Otherwise no special treatment is called for. If the hæmorrhage has been severe and there is a profound degree of anæmia, a blood transfusion might be called for. Some writers recommend the habitual use of iron, but in the average case the anæmia quickly disappears in response to the administration of the antiscorbutic.

¹ A. F. Hess, *Scurvy—Past and Present*, 1920, p. 238.

² H. Chick and M. Rhodes, *Lancet*, 7th December 1918, 774.

CHAPTER XI

RICKETS

FEW disorders of childhood are so important as rickets, because of its wide prevalence and the deformities and lowered resistance to disease that it causes, and especially because it is so eminently preventable, and, in its early stages, so easily cured. It used to be spoken of as a disease of the bones; but, while the alterations in the skeleton form its main peculiarities from the morbid anatomist's point of view, and as a matter of fact are still the most obvious and definite criteria for diagnosis, it must be appreciated that the tissues as a whole participate in the process, so that it is rather to be ranked as a disturbance of the general metabolism.

Frequency of Occurrence.—Rickets is one of the commonest diseases of childhood. It is a disease pre-eminently of urban and especially industrial urban areas, and of the poorer classes of the community, although it may be met with in rural districts and on occasion among the upper classes. Any estimate of the incidence of the disease will therefore depend on the nature of the particular area and on the class of the community investigated, and also, it should be remembered, whether chemical, histological, radiological, or clinical criteria are employed in the diagnosis. On clinical grounds alone the diagnosis in the early stages, and in the mild varieties of the disease, is very uncertain, so that the estimated frequency will vary according to whether only undoubted examples with deformities are classified as rachitic or early and doubtful cases are also included. Chemical and radiological investigations will detect examples that cannot be recognised clinically.

In Edinburgh between 1895 and 1900 it was found that rather more than 50 per cent. of the children under three years of age attending the out-patient departments of the Children's Hospital showed unmistakable clinical signs of rickets; and in Glasgow in 1917 it was estimated that well over 50 per cent.

of the same class of children had definitely suffered from the disease.¹ In 1897 Escherich, no doubt by the inclusion of milder examples, stated that 97 per cent. of infants between nine and fifteen months of age were rachitic. Schmorl,² in Dresden between 1901 and 1908, found during the post-mortem examination of 386 children under four years of age histological evidence of active or healed rickets in 89.4 per cent. In Newhaven, U.S.A., Eliot,³ as recently as 1925, on the ground of X-ray examination, put the incidence of rickets in the group of children investigated (one-third negro and two-thirds of Italian, Polish, Irish, and American stocks) at 90 to 96 per cent. Many of the cases were of course mild and evanescent, and were only recognised because of frequent examination throughout the whole year, but, as Eliot says, the "investigations have shown that a slight degree of rickets is well-nigh universal" among the poorer class children, both black and white, in Newhaven.

Although it is impossible to state with any degree of precision the incidence of the disease among the child population generally even in any one place, as it is influenced so much by race and social position, the above figures are sufficient to show how frequent a disease rickets is among city poor-class children. It may, however, be said that at least in England rickets in its severer forms is not so frequent as it formerly was. The severely deformed child is to-day a comparative rarity, and operations for bow-legs and contracted pelvis are exceptional. In the wards and out-patient departments of a Children's Hospital only slight and evanescent forms are usually encountered.

Geographical and Seasonal Incidence.—There is practically no region of the globe exempt from the disease. Modern investigations have shown that examples of the disease are to be found not only in the temperate zone but also in sub-tropical and sub-arctic regions. Although undoubtedly most prevalent in Europe and North America, the recent interest in the disease has disclosed its presence in India, South America and the West Indies, China and Japan, Australia and New Zealand, and in both North and South Africa, in all of which regions it had not been thought to occur.

¹ M. Ferguson and L. Findlay, *Med. Res. Coun. Spec. Rep.*, No. 20, 1917.

² *G. Ergeb. d. inn. Med. und Khde.*, 1909, iv., 403.

³ M. M. Eliot, *Journ. Amer. Med Assoc.*, 1925, lxxxv., 656.

A *seasonal incidence* in rickets has long been remarked upon by writers on the subject. It has always been observed that clinically it is most prevalent during the late winter and early spring, and that it is less common during the autumn. Hansemann stated that almost all children born during the autumn and dying during the following spring showed evidence of rickets. Hess expresses the same view thus: "The most

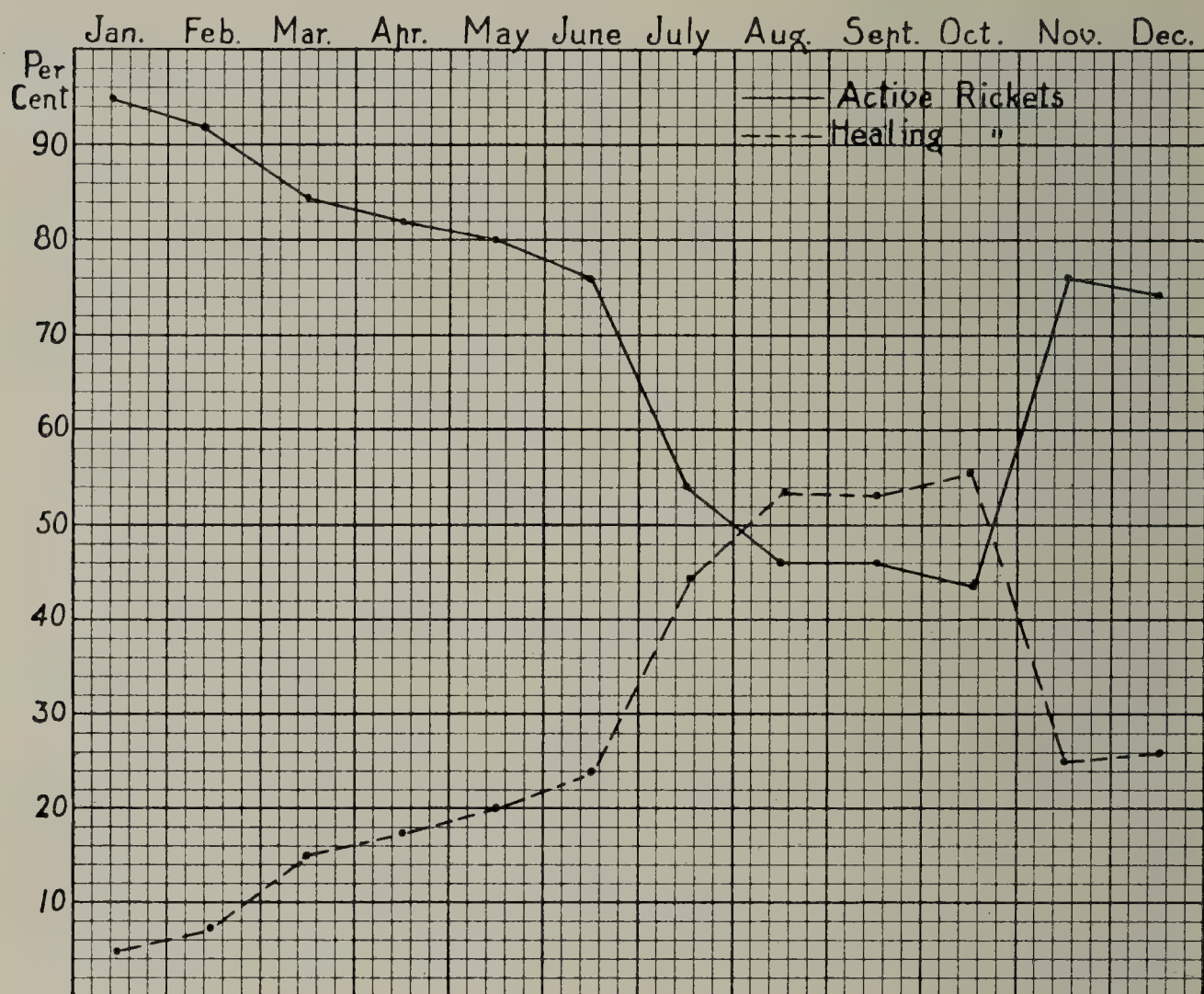


FIG. 76.—Showing percentage of cases examined post-mortem (386 in all) with active and healing rickets for each month of the year. (Compiled after Schmorl.)

inauspicious season to be born in respect of rickets is the early autumn." This is because the infants pass their most susceptible period during the most rickets-inducing time of the year.

The seasonal incidence of rickets is well exemplified in Fig. 76, which is compiled from Schmorl's findings and shows the incidence of active and healing rickets in the various months of the year.

Age of Onset.—Rickets is *par excellence* a disease of infancy. Various, however, are the expressions of opinion regarding the exact age at which it first makes its appearance. Just as has

been remarked of the incidence of the disease, any view of the age of onset will depend on whether metabolic, histological, radiological, or clinical criteria are employed.¹ Chemical or metabolic changes are always in advance of the radiological, and the radiological manifestations in point of time precede the clinical.

Clinically the condition is most frequently recognised between nine and twenty-four months of age, but on histological grounds Schmorl has shown that it is most frequently encountered, at least in the active stage, between four and eighteen months. Chemical or metabolic evidence would support that obtained by histology, and radiological evidence would put the range of incidence of onset as falling between that observed chemically and that observed clinically.

At one time it was considered that rickets might be of congenital origin. The older writers who expressed this view had confused rickets with congenital syphilis, achondroplasia, and osteogenesis imperfecta. This is not surprising as they based their conclusions on naked-eye examination of the bones. The characteristic histological picture of rickets was first described by Pommer in 1885.² Modern investigations based on metabolic, histological, or radiological evidence have failed to demonstrate congenital rickets.

Although, as already remarked, rickets is *par excellence* a disease of infancy and early childhood, it must be remembered that it may not only relapse during later childhood or adolescence, but also that it may develop in a previously healthy and non-rachitic individual as late as the twentieth year. When the disease sets in first after four years of age it is variously known as *late rickets*, *rachitis tarda*, or *juvenile rickets*. Rickets may also be met with as a complication of renal cirrhosis (so-called *renal rickets* or *renal dwarfism*), as a sequel to coeliac disease, and occasionally during vigorous thyroid treatment of cretinism. The relationship of these different varieties of the disease to one another will be discussed under the subject of pathogenesis.

Clinical Features.—The clinical manifestations may, for the sake of convenience of description, be divided into (*a*) symptoms, that is to say, what the mother has noticed about the child;

¹ Olive Macrae, *Archiv. Dis. in Child*, 1929, iv., 95.

² G. Pommer, *Osteomalacie und Rachitis*, Leipzig, 1885.

and (b) physical signs, which the medical man finds on examining the patient.

(a) **Symptoms.**—In most cases the first symptom which the mother notices is *excessive perspiration*. This is seen usually on the head, neck, and upper part of the chest, and less commonly all over the body. It is often so profuse that the sweat stands in beads on the forehead, and the pillow is so thoroughly wetted that it has to be frequently changed. The sweating is most severe when the child is sleeping, but it also occurs on slight exertion while he is awake; it often causes sudamina and miliaria. The amount of the perspiration of the scalp may cause undue anxiety on the part of the mother, who is apt to infer from it the presence of “water in the head”!

Another symptom which is often complained of—perhaps mostly in older infants—is great *restlessness* during sleep and a constant habit of throwing off the bed-clothes. Even when the weather is cold, the child will be found again and again with his bare legs and arms lying outside the blankets. At the same time he will roll his head from side to side on the pillow until the hair on the back of it becomes crumpled and thinned. Restlessness of this sort is not found only in rickety children, but it is much more common in them than in others.

Another thing which the mother often notices is the child's *disinclination to be moved*. His body and limbs seem tender, so that she can no longer use her accustomed freedom in washing and dressing him. He has to be gently handled, or he cries. Definite tenderness, however, is never due to rickets alone. It is much more likely to be caused by infantile scurvy, or by a local injury such as a fracture.

The child shows a great disinclination also to use his limbs. When he is left lying on the bed or sitting on the floor or on a chair, he keeps quite still, like an old man, and is not always on the move as a normal baby ought to be. When his feet are put to the ground, he raises them up and cries instead of trying to stand like a healthy child; and, if a relapse of rickets occurs after he has begun to walk, he may at once “go off his feet.” This disinclination for exercise is, however, not invariable. One of us (L. F.) has observed in each of a pair of twins equally severe radiographic rachitic changes, but whereas one child could not support himself erect, the other, as the mother expressed it, could “run like a hare.” (Figs. 77 and 78.)

Contrary to what is usually stated, rachitic children do not suffer more frequently from gastro-intestinal or pulmonary symptoms than non-rachitic children. In the severe examples of long standing such may occur, but it is probably more related to the hygienic conditions under which the children were living than to the disease itself. When there is much deformity of the chest, any pulmonary mischief contracted shows a tendency to persist and to become more severe. It must not be forgotten,

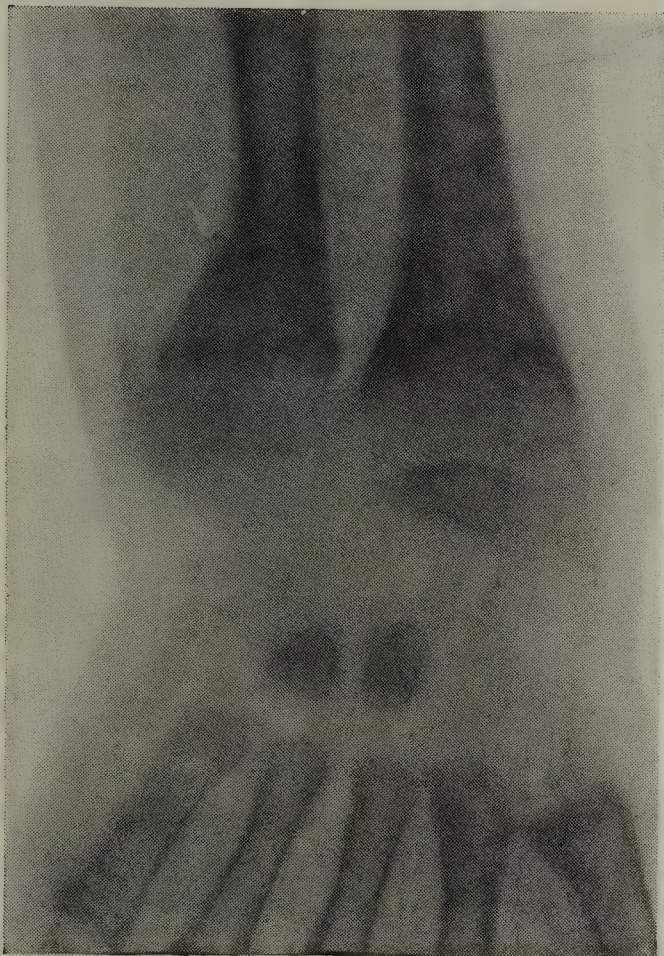


FIG. 77.—Skiagram of wrist showing marked Rickets in child aged $3\frac{1}{2}$ years (twin sister of case shown in Fig. 78). This child was quite unable to walk.



FIG. 78.—Skiagram of wrist in child aged $3\frac{1}{2}$ years (twin brother of case shown in Fig. 77) showing marked Rickets but yet could run about.

however, that the child may have been confined to the house on account of gastro-intestinal troubles, or a tendency to bronchitis, and that in this way the rachitic mischief has come about.

A history of attacks of laryngismus stridulus, convulsions, or carpo-pedal spasm may be obtained.

(b) **Physical Signs.**—One of the most striking points about the rickety child is that he is flabby. He may or may not be thin, often he is unusually fat. His muscles are soft and he is languid in his movements and easily tired. He may look pale,

but except in complicated cases there is no anæmia¹; in fact, the hæmoglobin and red-cell contents may be above the normal.

No effect on the child's growth may be noticeable during the early stage of the disease, but if the rickets has been of some duration and is severe, and especially if it is so at an early period, then there is, sooner or later, some stunting of the growth. Rickets is the most frequent cause of the growth of the city child being arrested.

The temperature is not raised. If fever is present, it is not the result of the rickets, but indicates the presence of some complication.

The outline of the head may not be altered in the early stages or in slight cases; but, when the disease has lasted for any time, it generally shows a characteristic *square shape* with an expansive overhanging forehead (Figs. 38, 39, and 79 to 84). The *fontanelle* is almost invariably *larger than normal*, and it may be found widely open at the end of the second, or even in the third or fourth year. Its margins also are abnormally thin and yielding. The coronal and other sutures often gape a little, and their margins are soft and pliant like those of the fontanelle. This softening of the cranial bones (*craniotabes*) may involve the centres of the flat bones, especially the occipital and parietal, so that the bone can be indented with the finger just as the lid of a thin metal box. Craniotabes is often the earliest evidence of the disease and may be quite marked in infants a few months old when as yet no epiphyseal changes are apparent. It is important not to mistake congenital islands of defective ossification for true rachitic craniotabes, which is never present during the first weeks of life. In addition to thinning of the cranial bones, areas of thickening (*bossing*) may make their appearance. This bossing is usually present over the parietal and frontal eminences, contributes towards the altered shape of the head, and usually only makes its appearance during the later months of infancy (Fig. 40, p. 69).

The jaws are often noticeably affected, and in time may become considerably altered in shape (Fleischmann).² The upper is laterally compressed in front so as to form a sort of beak, while towards the back its alveoli are far apart and turn somewhat outwards. The lower jaw becomes angular instead

¹ L. Findlay, *Lancet*, 1909, i., 1164.

² *Klinik der Pädiatrik*, Bd. ii., Vienna, 1877.



FIG. 79.—Early Rickets in boy of 21 months. Commencing affection of cranium, thorax, abdomen, and epiphyses.

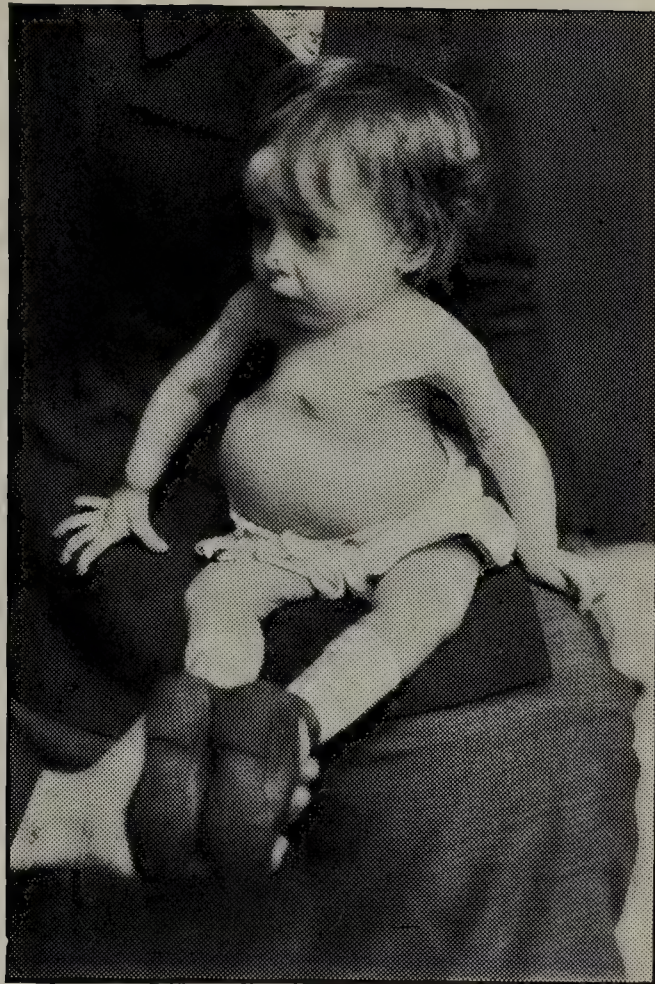


FIG. 80.—Severe Rickets in a girl of 2½ years. Deformed thorax, large abdomen, slight curvature of arms, laxity of ligaments in lower extremities—the feet being turned backwards without the child seeming to notice it.

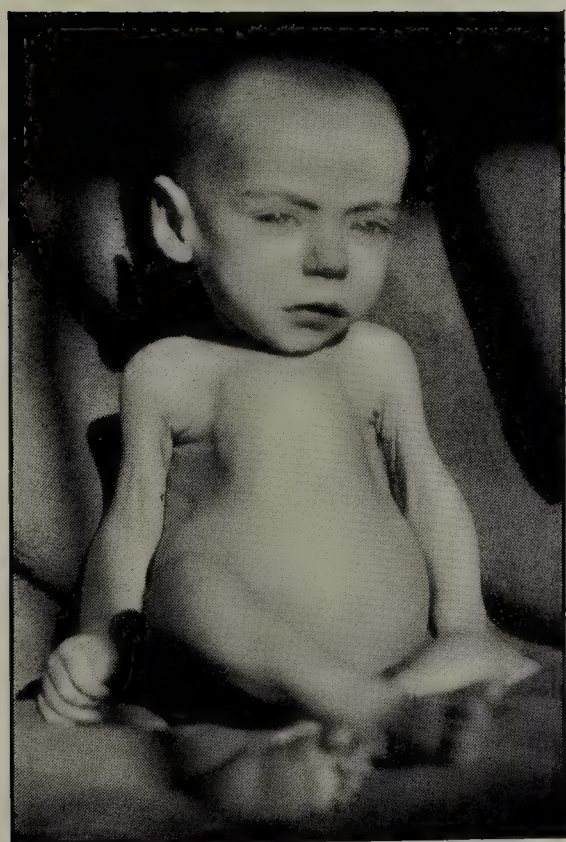


FIG. 81.—Rickets, with severe affection of head and thorax.



FIG. 82.—Rickets, with severe affection of head and thorax.

of rounded in outline, the front being flat with angles situated at the canine teeth, and its alveolar margin tends to turn inwards. One result of this change in the form of the jaws and in the direction of the alveoli is to prevent the proper apposition of the upper and lower sets of teeth.

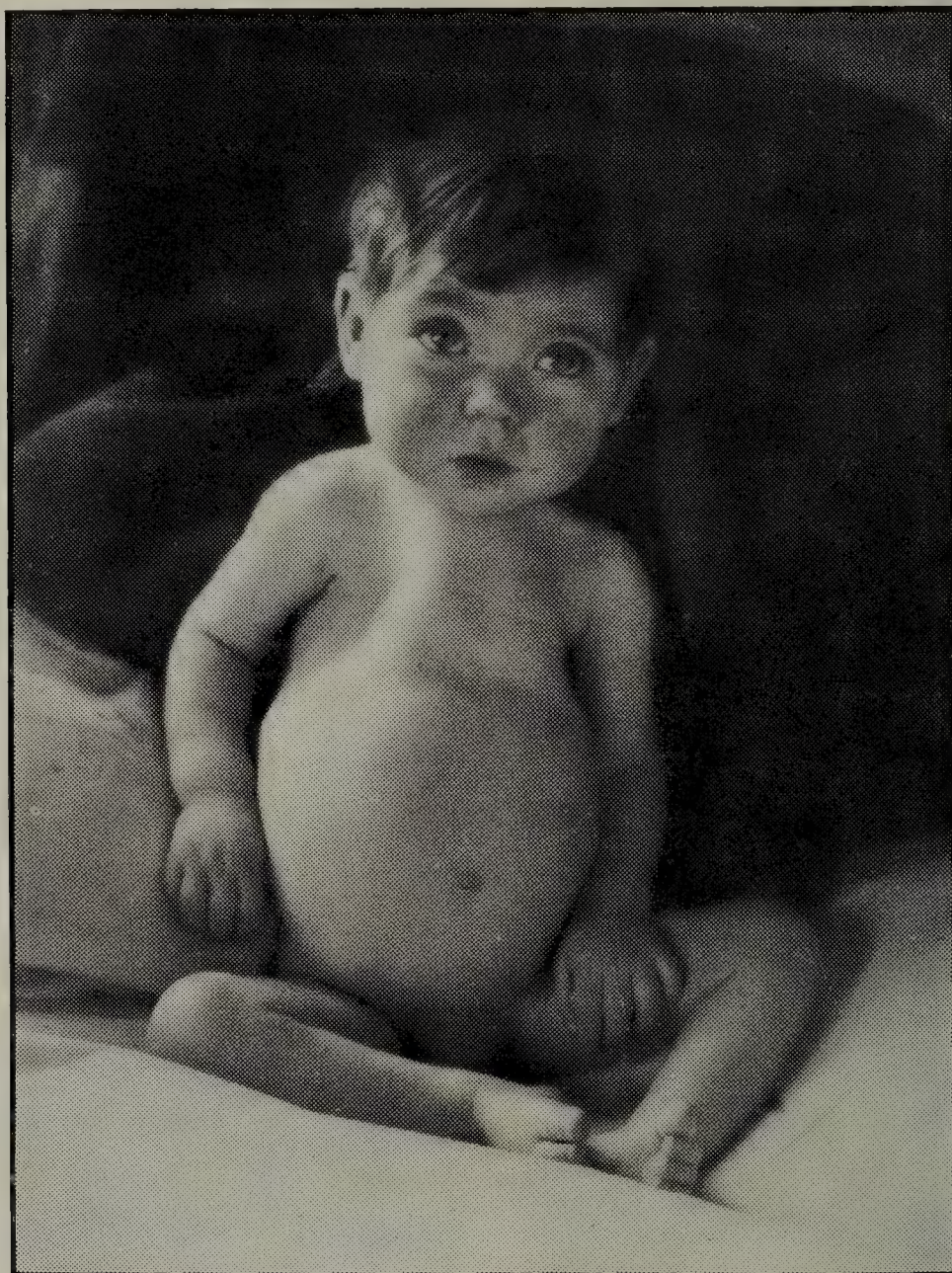


FIG. 83.—Rickets, with severe affection of head and thorax, in a girl of 2 years.

Dentition is almost always *delayed* beyond the normal time, and the intervals between the appearance of the teeth vary greatly in duration. The teeth are also apt to come *in the wrong order*, and to appear one by one, instead of in pairs as usual. In long-standing cases some of the incisors often drop out.

The effect of rickets on the chest wall is seen in *beading of the ribs* (enlargement of the costo-chondral junctions) and in various degrees of *thoracic deformity*; these are considered

later (Chapter XXV). *Rickety curvature of the spine*, and its diagnosis from Pott's disease, have been already referred to (p. 78). The curvature begins early in the disease and soon becomes severe if the child is allowed to sit up too much (Figs. 50 and 84). This is due in great part to softening of the ligaments.

The changes in the bones consist in enlargement at the junction of the epiphyses and diaphyses with softening and bending, and occasional fractures of the shafts. The *enlargement at the epiphyses* corresponds to the beading of the ribs, and may be found beginning by the third or fourth month, although it does not attain any great size until the child is old enough to use the affected limbs more actively. It is generally first and most clearly seen at the lower end of the radius. In the lower limbs, the distal ends of the tibiæ are the parts where the first and greatest enlargement usually occurs. The legs are affected later than the arms, and the epiphyseal swellings of the leg-bones do not usually attain a considerable size except in children who are trying to walk.

The *bending of the long bones* depends on the amount and direction of the pressure to which they are subjected. It does not occur to any extent in infants who are kept lying flat. Bending of the arms (Fig. 80) is commonly met with in severe cases and is secondary to kyphosis. The child assumes a frog-like posture, in which he tries to relieve his weak back muscles by bearing the weight of his head and shoulders as much as possible on his arms.

Bow-leg and coxa vara are commonly caused by the child standing and walking while the bones are too soft to bear the weight of the body. The former is more apt to take place in those children whose muscles and ligaments are not very much weakened by the disease. When those structures are much softened, knock-knee with flat-foot is more likely to result. Antero-posterior bending of the femora occurs when the

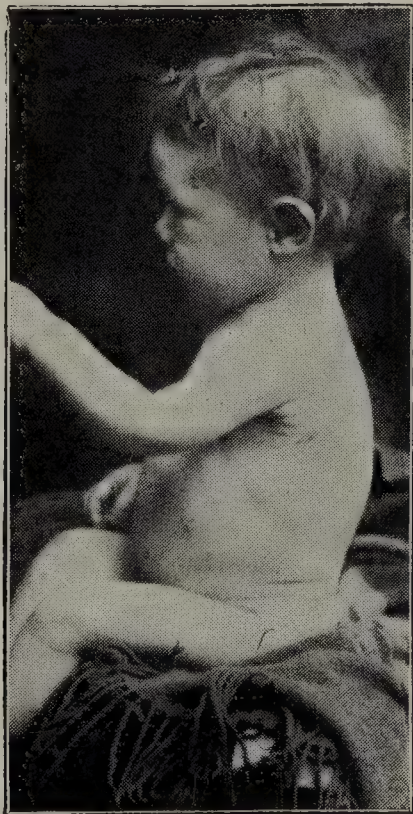


FIG. 84.—Rickets in a boy of 20 months. Square head, beading of ribs and spinal curvature.

children have been carried much on the arm, or been used to sitting long on a chair with their legs hanging down; and a similar backward bending of the lower part of the tibia and fibula may occur if the child habitually sits with one leg laid over the other and the foot unsupported (Fig. 325, p. 932).

Fractures of any of the long bones sometimes occur (Figs. 85 and 86). In some cases they are multiple, and they require very little force to produce them. Fractures of the ribs are caused



FIGS. 85 and 86.—Rickets in a girl of 6 years. Badly united fractures of clavicles, right humerus, left radius and ulna, and of ribs below the right scapula.

by lateral compression of the chest with the hands, in lifting the child; those of the clavicle and humerus, by lifting him by the upper arms. The bones of the forearm are sometimes bent and fractured owing to the child's suddenly tripping and falling while he is being encouraged to walk with the mother's hand firmly grasping his wrist. Under these circumstances, as the parent is right-handed, it is usually the child's left arm which is fractured (Fig. 85). Fractures of the leg are not common, but those of the thigh are often met with; they may be due to a fall from a chair, or other slight accident. When a fracture occurs, nothing may be noticed but local pain on movement and on

pressure; and the nature of the injury may not be recognised until callus begins to form.

The peculiar conformation of the hands and fingers which is often seen in the progressive stage has been already described (Fig. 57, p. 93).

The bones most affected vary in different cases. This may be partly explained by the hypothesis that rickets *tends to affect those bones which are in process of the most active development* (Baginsky).¹ Thus we find the cranium most severely affected in young babies, the thorax, vertebræ, and arms in those a little older, and the lower limbs in those children in whom the process is late of developing. It would appear that *the degree of swelling present at the epiphyseal ends of any of the long bones is directly proportionate to the amount they are being moved*. Hence they are always small when the muscles are too feeble to allow much movement of the limb, and large in children with stronger muscles who walk much or make much use of their hands. Similarly, there is never any great beading of the 1st or 2nd ribs which move little, and always a great deal of the 5th and 6th which have a wide range of movement. One of us (L. F.), in a puppy in whom rickets had been induced, observed a complete absence of epiphyseal changes in one hind leg which had been the seat of a fracture and had been kept more or less at rest (Fig. 87).²

Radiographic Signs of Rickets.—As previously mentioned, clinical signs of rickets may be exceedingly uncertain. If it is a frequent experience to be unable to diagnose with certainty the presence of rickets, it is a still more frequent experience to be unable to state even in the presence of marked lesions whether the disease is active or healing. For this purpose either radiographic or metabolic evidence is necessary.

In the *active stage* of the disease the epiphyseal extremity of the diaphysis in the radiogram has lost its definition, and instead of having a sharply defined and horizontal or slightly convex end, it is fuzzy and slightly or distinctly concave. The epiphysis, not being calcified at this age, probably does not show. With an increase in severity the area of decalcification extends,

¹ *Practische Beiträge zur Kinderheilkunde*, H. ii. Rachitis, Tübingen, 1872, 50.

² M. Ferguson and L. Findlay, *Medical Research Council Report*, No. 20, 1917, p. 29.

the end of the bone becomes enlarged and has a deep cup-shaped distal extremity which gradually fades in density or may show definite serrations. The diaphysis generally in its want of density shows decalcification with a blurring of the normal trabecular markings, and in the younger children

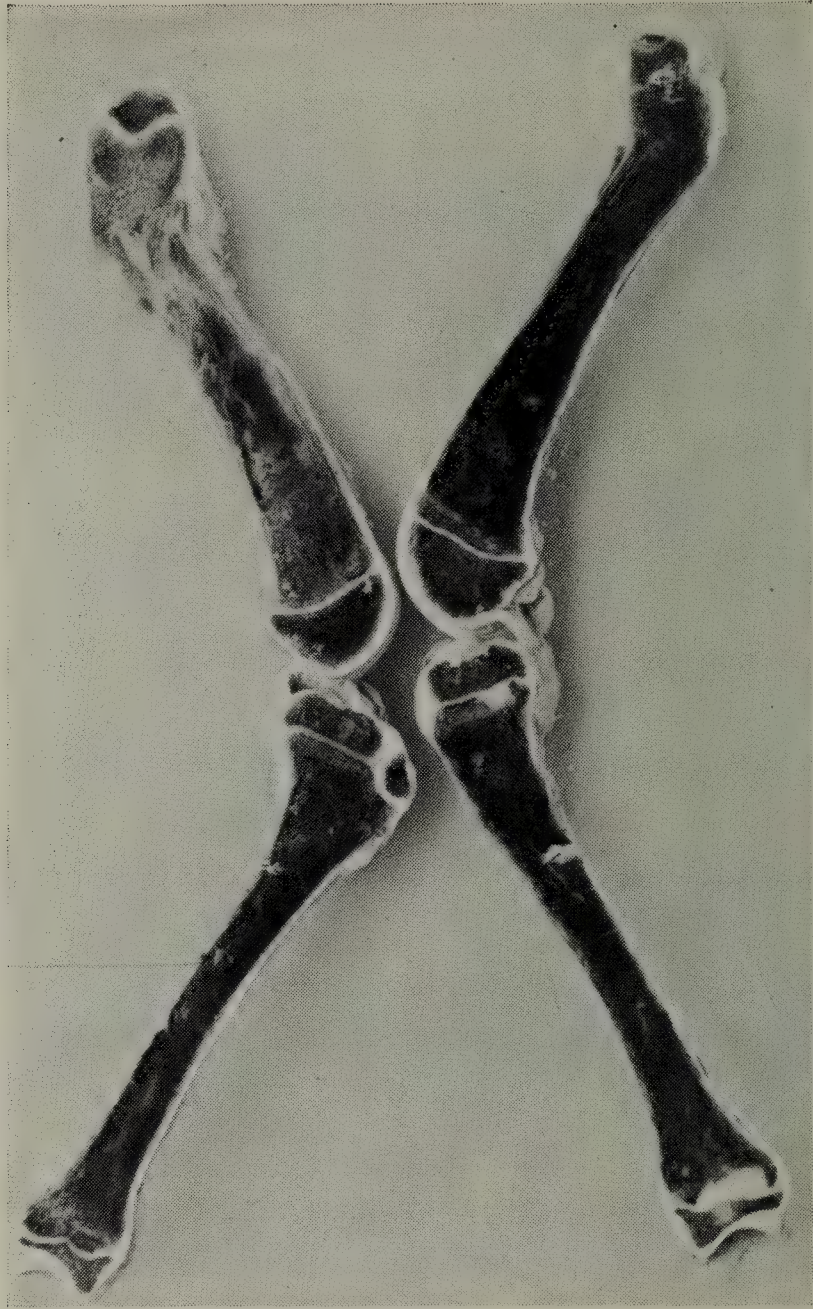


FIG. 87.—Bones of hind legs in case of experimental rickets in the dog showing rachitic changes in one leg and none in the other, which is the seat of a fracture of the femur.

definite and poorly calcified periosteal thickening (Fig. 88). Unsuspected fractures showing little displacement and callus formation may be observed (Fig. 96, p. 281).

When *healing* sets in the first change is a return to definition of the extremity. This line gradually becomes more marked and calcification extends diaphyseally as well as epiphyseally until the normal contour of the bone is regained. At the same

time the bone generally increases in density, and the subperiosteal thickening becomes properly calcified and is indistinguishable from the rest of the shaft (Fig. 89).

It is important to remember that after healing has commenced it will not be observed in the radiogram for a matter of three weeks. This shows us that an isolated radiogram is not of any value in estimating the activity of the condition. The bones may appear the seat of florid rickets and yet healing is in progress, and the bones may appear quite normal although the disease has already developed. It has been our practice to insist on an observation period of at least three weeks before

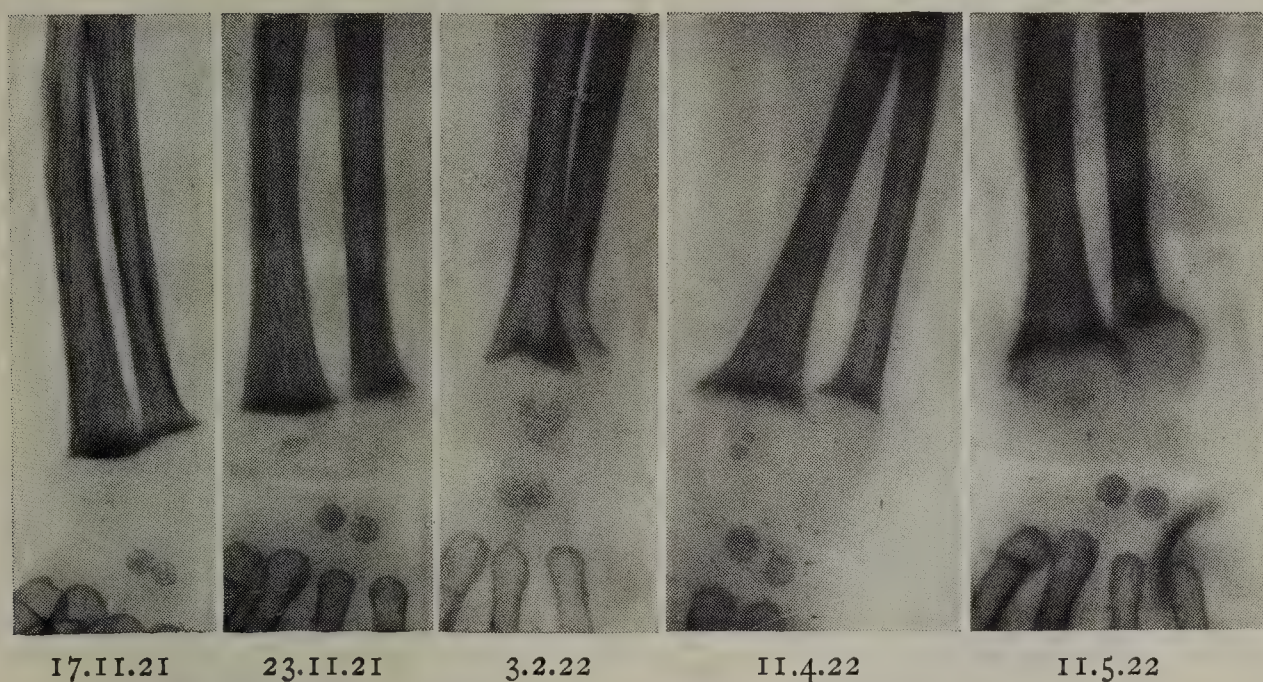


FIG. 88.—Series of skiagrams in a child (born June 1921) showing the development of Rickets. The dates on which each examination was made are recorded.

coming to a decision on this matter. After healing has commenced it is usually complete within eight weeks. These various points are illustrated in the accompanying radiograms (Figs. 89 and 90). Ultimately no trace of the disease may be observed on examination of the bones. Transverse striation of the epiphyseal end of the diaphysis is sometimes stated to be evidence of old rickets. This is, however, not the case. These transverse lines are frequently observed in children who have never suffered from rickets. They are evidence of interrupted growth due, in some instances, to an intercurrent illness and are thus analogous to the transverse grooving of the nails (p. 412). The only definite evidence of past rickets is Macewen's tibial spine (Fig. 93, p. 276).

Extreme *laxity and extensibility of the ligaments* are striking

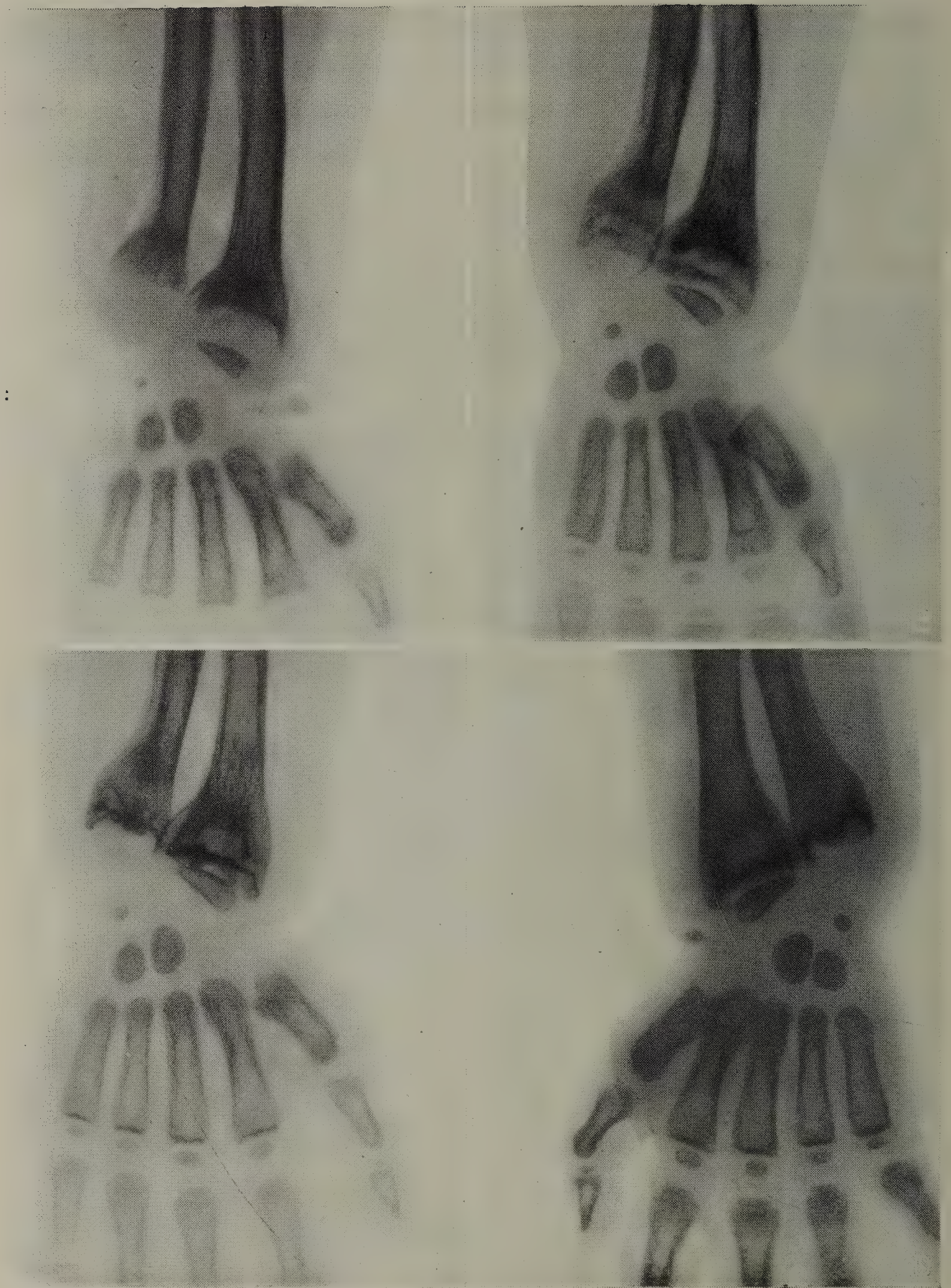


FIG. 89.—Series of radiograms taken before and during the administration of ultra-violet rays. (A) 24.10.27. Note cup-shaped thickened ends of radius and ulna with poorly calcified subperiosteal bone. (B) 2.12.27. (Ultra-violet ray therapy was commenced on 15.11.27.) Note calcification of most recently formed bone—extreme end of diaphysis. (C) 10.12.27. Note further stage of healing, calcification being apparent throughout diaphysis. The poorly calcified periosteal bone is well seen here. (D) 30.12.27. Almost completely healed. The whole bone is denser, the periosteal bone is hardly to be differentiated, the epiphysis of radius is also much denser and more uniform, and excessive calcification of the most recently formed bone is well marked. (Girl aged 2 years.)

features in many cases of rickets and are most characteristic of the disease. Their softness contributes largely towards the

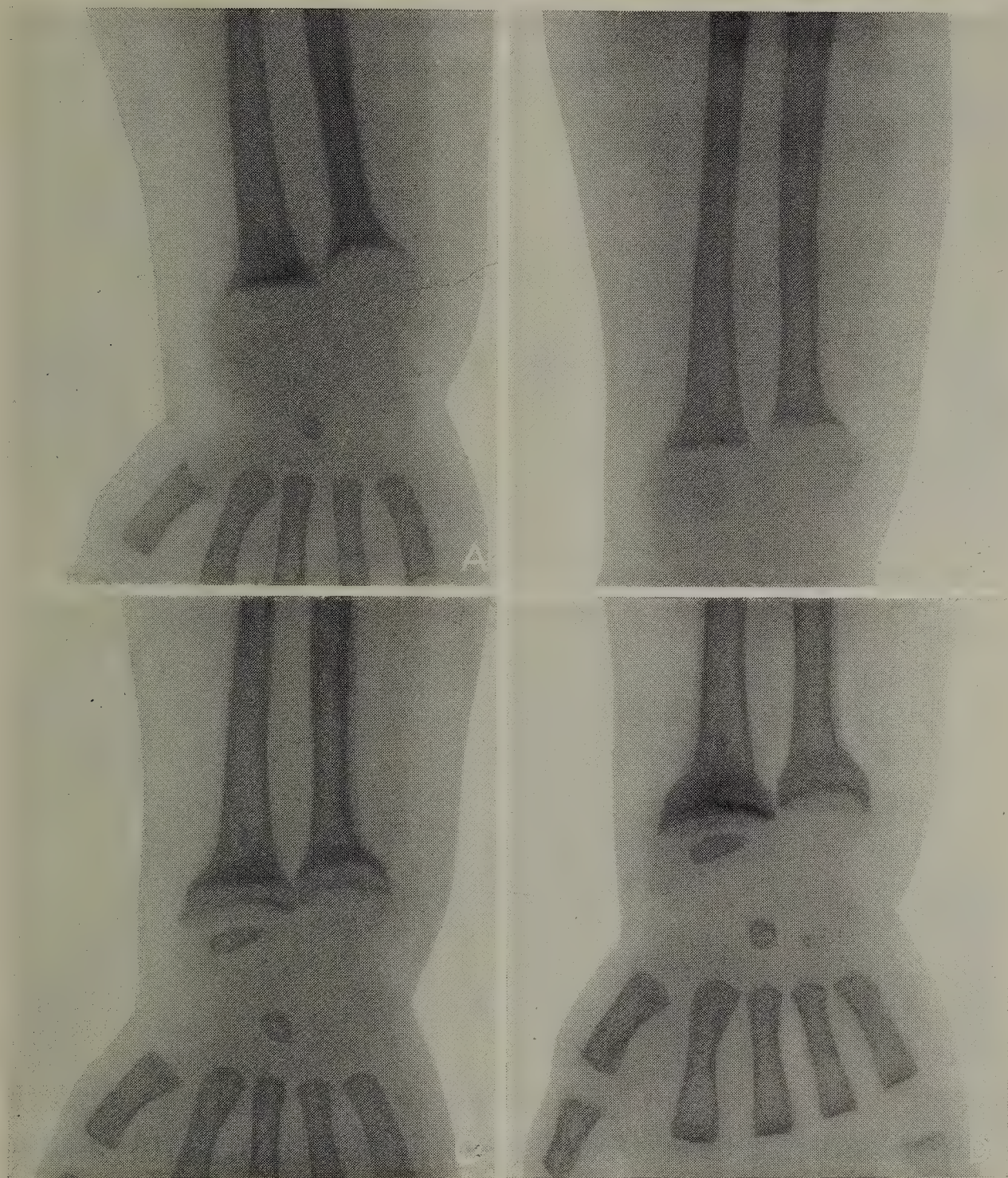


FIG. 90.—Series of skiagrams in case of Rickets before and during the administration of cod-liver oil (boy aged 1 year and 2 months). (A) 1.10.24. Note marked cupping of ends of radius and ulna. (B) 31.10.24. Note absence of any change in condition—child had been confined to bed in hospital ward on ordinary diet. Cod-liver oil 5i five times daily commenced on this date. (C) 18.11.24. Note extensive healing which has taken place during course of 18 days, evidenced by line of calcification at level of most recent bone formation with increased calcification of shaft. (D) 25.11.24. Note that healing is further advanced.

deformity in rickety spinal curvature and bending of the limbs ; it often also leads to flat-foot and knock-knee. When it is present to a considerable degree, the limbs can be twisted about

to a surprising extent and the feet turned with the toes pointing directly backwards without inconveniencing the child (Fig. 80). When antirachitic treatment is successfully employed this laxity of the ligaments very rapidly diminishes.

Weakness of the muscles is as characteristic of rickets as bending of the bones.¹ It may be severe in cases where the latter are apparently but slightly affected. When this is so, the condition is sometimes spoken of as rachitic pseudo-paralysis.

The *abdomen* in rickety children is always protuberant. This is owing chiefly to lack of tone in the muscles of the abdominal wall and intestine, but also is contributed to by dyspepsia, which may accompany the disease and cause an accumulation of flatus. It looks even larger than it is, owing to contrast with the narrowed chest. In many cases the flatulent distension leads to diastasis of the recti muscles.

So far as the *liver* and *spleen* are concerned there is no evidence that they suffer in rickets. Both organs may be palpable. This is not evidence of enlargement, and is most probably due to displacement downwards following contracture of the thorax. The liver is so easily palpable in the infant and young child that it is difficult to estimate clinically slight degrees of enlargement. If the spleen is definitely enlarged it is invariably due to some complication, *e.g.*, syphilis, tuberculosis, anæmia, or a chronic pyogenic infection.²

Enlargement of *lymph nodes* and *tonsils* with the presence of adenoids has been considered characteristic of rickets. Palpable glands are so common in the class of child subject to rickets that no significance can be attached to their presence, and the frequency with which tonsillar enlargement is present in non-rachitic children must dispose of any association between these two conditions.

The urine shows no constant abnormalities discoverable by the ordinary clinical tests.

As previously mentioned (p. 249), although the rachitic child may appear pale there is no anæmia.³ The proportion of hæmoglobin and the number of red cells are if anything raised; the leucocytes may be normal or slightly increased in number,

¹ Douglas Galbraith, *Brit. Journ. Child. Dis.*, 1923, xx., 143.

² J. M. Cowan and J. C. M'Clure, *Brit. Journ. Child. Dis.*, 1906, iii., 343.

³ L. Findlay, *Lancet*, 1909, i., 1164.

but otherwise they present no abnormality. Definite blood changes can be taken as evidence of some complication.

In a disease characterised by a deficiency of calcium in the bones it is only natural that estimations of the calcium content of the blood should have been carried out. This in uncomplicated rickets was, however, invariably found within normal limits, which amounts to between 10 and 11 mgrm. of CaO per cent. When tetany complicates rickets the calcium content is lowered and may give readings of only 3 mgrm. per cent. On this account it was customary for some writers (Shipley, Park, McCollum, and Simmonds¹) to speak of calcium-poor and calcium-rich rickets. In 1919 Iversen and Lenstrup² discovered that in rickets the inorganic phosphorus content of the blood, normally varying between 4 and 5 mgrm. per cent., is decreased. This finding was soon afterwards confirmed by Howland and Kramer³ and is now recognised as one of the most constant and delicate tests for the presence of active rachitic mischief.

Howland and Kramer have attached great significance to the proportion of phosphorus and calcium for normal ossification. According to them, when the product of the number of mgrm. per cent. of the serum inorganic phosphorus and calcium amounts to 40 or more, then rickets is either absent or healing, but when the product is 30 or less, the process of normal ossification is in abeyance and rickets is developing. Although the low phosphorus content of the blood and a product of the calcium and phosphorus less than 30 is true of the average case, the overlap between normal and abnormal is so great that these factors should not be given too much significance.

Nervous manifestations are not infrequent in rickets. These are almost entirely of the nature of tetany. It is difficult to express the true relationship between rickets and tetany. In both there is an upset of the calcium metabolism, and it may be that the one makes the child susceptible to the other. This question will be discussed in detail when dealing with tetany (p. 546). The restlessness and profuse perspiration during the early stages of the disease are also probably of a nervous nature. It is frequently stated that rickets affects adversely the mental

¹ P. G. Shipley, E. A. Park, E. V. McCollum, and N. Simmonds, *Amer. Journ. Dis. Child.*, 1922, xxiii., 91.

² P. Iversen and E. Lenstrup, *Hospitals Tidende*, 1919, lxii., 1079.

³ J. Howland and B. Kramer, *Amer. Journ. Dis. Child.*, 1921, xxii., 105.

condition of the child. In our experience there has been little evidence of any such occurrence. The decision of this question is beset with many difficulties as mental tests, which alone would reveal the slight deficiencies, which only can be present, are not possible under three years of age, when the disease is most frequent, and it has never been held that the defect of intelligence is permanent, so that examination of the older child is of no value.

Metabolic Changes in Rickets.—Although it has been definitely proved that rickets cannot be induced in the animal by a diet poor in calcium, and it is apparent to every pædiatrician that the disease develops when a child is receiving more than the necessary amount of this element, as, *e.g.*, when the diet consists of undiluted cow's milk, the most constant change observed during the course of the disease is a diminished utilisation of lime. Instead of the infant retaining, as in health, 0.1 gramme CaO per kilo body weight per day, the amount retained may be only 0.01 gramme or less per kilo per day. Some authors record that even more lime may be excreted than is ingested, *i.e.*, there results what is called a "negative balance."

As the excessive excretion of calcium is in the fæces, the urinary excretion being diminished, it has been much debated whether this state of matters results from diminished absorption from the bowel or from an excretion by the bowel of some already absorbed but not utilised. The fact that the amount normally excreted by the kidney is markedly lessened would tend to make one ascribe the defect to one of absorption, and this is the view generally held. The normal blood calcium, on the other hand, does not suggest defective absorption. It must be remembered, however, that a low blood calcium is fraught with danger of serious phenomena (tetany), and thus it is possible that Nature will do all in her power to hinder such an occurrence. The necessary amount of calcium required for this purpose is infinitesimal in comparison with the total amount in the body. In the new-born infant the total calcium content (CaO) of the body is 28 grammes, whereas that of the blood is at most 0.02 grammes.

It is of course impossible from an examination of the fæces to decide what proportion of the lime is simply the unabsorbed residue of that present in the food, and the proportion which has been absorbed and later excreted. Some workers doubt if

lime is ever excreted by the bowel, at least to any serious extent, and work which has been carried out to elucidate the question has given discordant results. Telfer,¹ by cutting down the lime in the diet to a minimum, finds that it practically disappears from the fæces, and Macrae, in experiments with the isolated loop of gut in the pig, has seen little evidence of excretion by the bowel. Grosser,² on the other hand, after intravenous injections of salts of lime, has found more calcium in the fæces than was given *per os*. It is of course possible that although there may be a minimal excretion of lime by the bowel in health, a perversion of the process may occur in disease. In fact, Ford³ has observed in a child suffering from diabetes more lime over a seven-day period in the fæces than could be accounted for by that ingested, and Morris and MacRae⁴ have shown that the same may result during acidosis induced by ammonium chloride. But in both of these conditions there is, in marked contrast to what prevails in rickets, an increased excretion by the urine as well.

Since most of the phosphorus of the body is in combination with lime in the bones, it is readily understandable that the behaviour of the metabolism of lime and phosphorus should be parallel. However, instead of the loss of the phosphorus, as is the case with the lime, being invariably by the bowel, it may take place by either the bowel or kidney. Whether or not phosphorus is excreted by the kidney depends on whether the conditions in the gut are favourable for its absorption.^{5, 6} If there is an excess of lime available for combination with phosphorus then the insoluble calcium phosphate results and is excreted *via* the fæces, whereas if there is a diminished amount of calcium in the gut, or at least of calcium available for combination with phosphorus, the phosphorus is absorbed and excreted *via* the kidney. This fact that phosphorus may be excreted in excess by the kidney, which never happens with calcium, is a strong argument in favour of the theory of

¹ S. V. Telfer, *Quart. Journ. Med.*, 1924, xvii., 245 ; L. Findlay, *Journ. Amer. Med. Assoc.*, 1924, lxxxiii., 1473.

² P. Grosser, *Zeit. f. Kinderheil.*, 1920, xxv., 141.

³ F. J. Ford (unpublished).

⁴ N. Morris and O. MacRae, *Arch. Dis. Child.*, 1930, v., 207.

⁵ S. V. Telfer, *Quart. Journ. Med.*, 1922-23, xvi., 45 and 63, and 1923-24, xvii., 245.

⁶ G. Murdoch, *Arch. Dis. Child.*, 1927, ii., 285.

defective absorption of lime being the underlying cause of the metabolic change.

Pathology of Rickets.—We owe our knowledge of the pathology of rickets to the classical studies of Pommer which were published in 1885. Until this time there existed much confusion. Prior to Pommer, congenital syphilis, achondroplasia, and osteogenesis imperfecta were mistaken for rickets, and

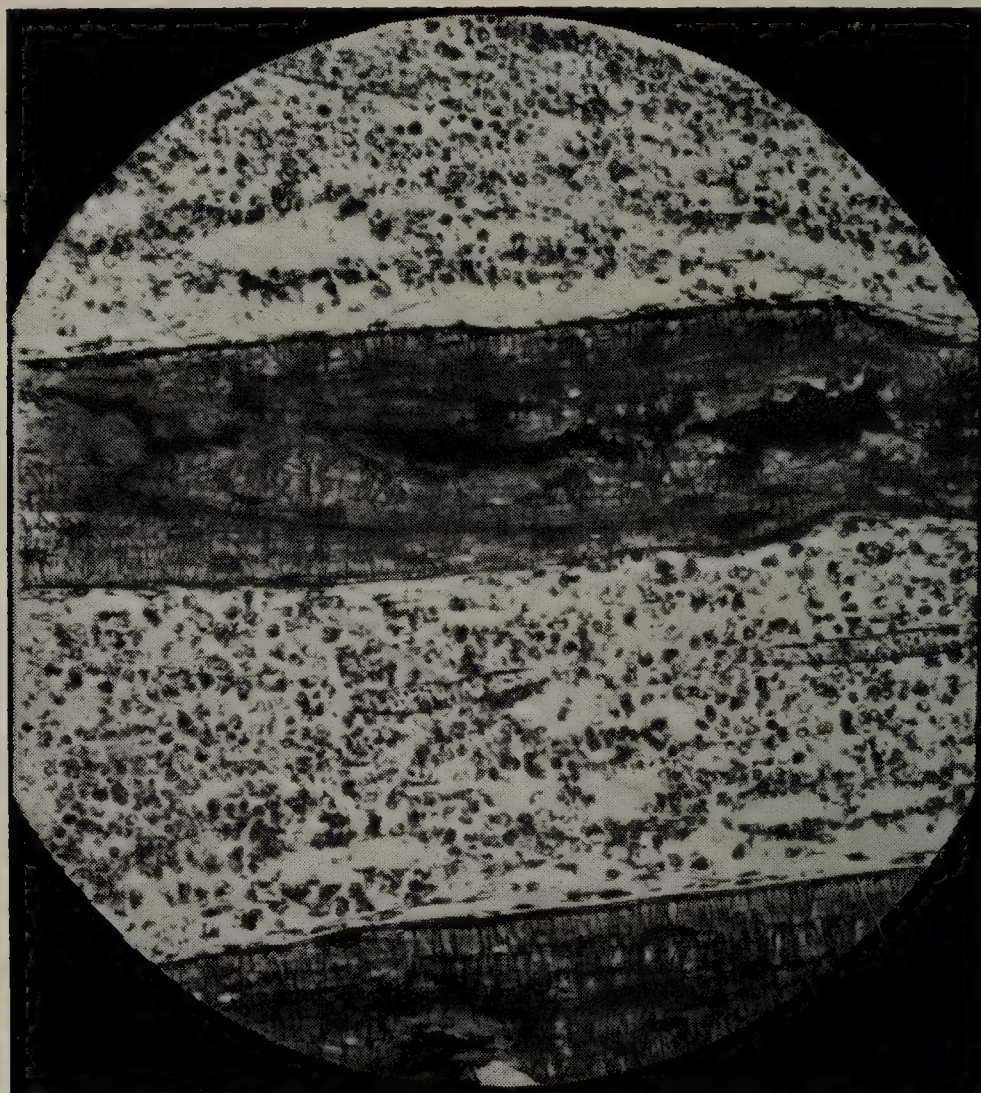


FIG. 91.—Section showing structure of trabeculae of normal bone.

engendered the idea that it might be of a congenital nature, and in the experimental animal softness of the bones and osteoporosis were considered evidence of rickets.

Fundamentally, rickets is characterised by an abnormality of growing bone. There is an increased attempt at the formation of bone which, however, falls short of the completed process, so that, instead of normal osseous tissue, so-called osteoid tissue is formed. The cartilaginous or membranous framework does not, or only very incompletely, become calcified. It is this exuberance of growth of cartilage which gives rise to the thickenings which are especially marked at the growing ends of

the bones. Normally the bony trabeculæ are almost wholly occupied by parallel rows of uniformly sized bone-cells, with their processes running practically to the margin and leaving only the merest rind of structureless tissue (Fig. 91). In rickets, on the other hand, the trabeculæ persist for the most part as homogeneous processes with or without ghosts of bone-cells (the so-called osteoid tissue) with small central zones

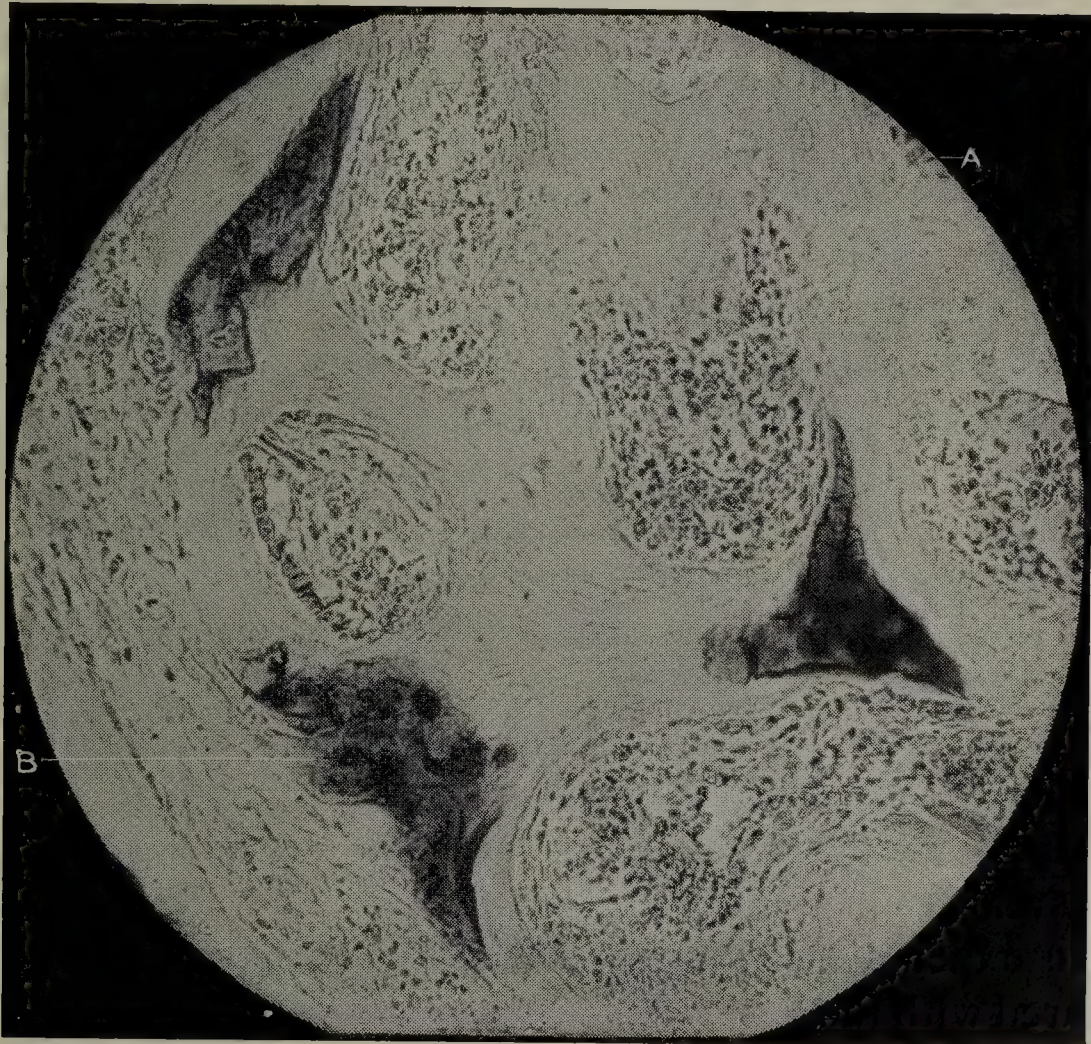


FIG. 92.—Section of rachitic bone showing osteoid tissue (A) and poorly formed and irregularly placed bone-cells (B). (Experimental Rickets.)

occupied by dwarfed and irregularly placed bone-cells (Fig. 92). *It is the development of this osteoid tissue which is the characteristic histological feature of rickets.*

It is the exuberance of growth of cartilage which gives rise to the thickenings which are specially marked at the growing ends of the bones, and it is the absence of calcification which renders the bones soft and liable to bending and is responsible for the radiographical abnormalities.

Pathogenesis. — For many years, as we have already remarked, it has been much discussed whether the absence of calcification of the bones is in consequence of an inability of

the growing cartilage to absorb or fix the calcium which is brought to it by the blood in sufficient amount, or whether it is because an ample amount of calcium is not available for the process. The possibility of this calcium starvation being dependent either on a deficient quantity in the diet or on defective absorption from the bowel, even although ample is present in the diet, has been much debated. The fact, however, that in the preliminary stage of the rachitic mischief the growth of cartilage is hyperplastic and erratic, points to it being a disease of the whole process of ossification, and not one simply of the supply of either calcium or phosphorus. In some way the regulating mechanism of ossification is upset. Although we are still ignorant of the true disturber of this process, much has been learned within recent years regarding factors which influence it and these will be discussed under etiology.

Etiology.—Than rickets no disease has within recent years been more exhaustively studied, and yet we are ignorant of its real etiology. But we have learned, as just stated, about many factors which definitely influence ossification.

In the first undoubted description of rickets by Soranus Ephesius,¹ a physician resident in Rome during the first and second centuries of the Christian era, various hypotheses are suggested, but this sage physician summed up the question in the remark that “the matter lies in inexperience with regard to the rearing of children,” and he drew attention to the lack of care expended on the children by the Roman mothers as compared with those of Greece, where the disease was apparently less common. To-day, nearly two thousand years later, the same strictures regarding the parents of rachitic children are equally applicable, for, even although the real cause remains a mystery, there is nothing more certain than that the obedience to the ordinary laws of health will hinder the development of rickets.

In the writings of the Middle Ages, and indeed well into the nineteenth century, almost every conceivable factor in the child's up-bringing was blamed. Glisson² included in his long list of primary or secondary causes everything that could be considered

¹ Soranus Ephesius, *Dietz Text*, 1838. (German translation by H. Luneberg, *Die Gynaäkologie*, Munich, 1894.)

² Francis Glisson, *De Rachitide sive Morbo Puerili, qui vulgo The Rickets dicitur*, London, 1650.

to render the parents less likely to produce a healthy stock, or weaken the child after birth and cause what he calls cold and distemper, and so modify the nutriment of the blood, which state for him was the essence of the disease. In the main, however, the causes have been assigned to errors in diet and environment, although, with the birth of bacteriology, and at a later date of endocrinology, there were periods when a bacterial factor and a glandular factor were sought for and accepted. At the present time, when deficiency diseases are the fashion, a theory which involves the conception of the absence of some vital and potent principle is the one adopted by most workers. In connection with each of these various theories there has always been one or more disturbing and anomalous fact which hindered it from completely filling the bill. As the result of the enormous amount of exact clinical and experimental work of the last twenty years we have learned, however, to appreciate certain factors which influence ossification and predispose to the development of rickets.

Congenital Factors.—As is the case with most diseases there is undoubtedly a varying susceptibility to rickets. This is shown by the fact that children fed, housed, and reared under identical conditions do not suffer equally. On what this relative immunity depends is unknown: there is no proof that the child is born with a store of any protective substance. There is, however, ample evidence of the child who is born with an insufficient store of calcium being specially susceptible to the disease. Most of the calcium in the foetus is deposited during the last two months of intra-uterine life, and hence the degree of maturity of the new-born is a factor of first importance. Indeed, the premature infant almost invariably develops the disease to some extent.

There is no evidence that hereditary factors play any part in the causation, or that the children of rachitic parents are specially prone to the disease. Any supposed evidence in this direction can usually be explained by a persistence of the bad hygienic conditions under which the parents had been reared, or by the effect of the health of the mother on the same. Undoubtedly the health of the mother may modify the general care of the child, but there is no evidence of any paternal or maternal factor otherwise playing a part. Telfer¹ has shown

¹ S. V. Telfer, *Glas. Med. Journ.*, 1930, cxiii., 246.

that the milk of the badly-nourished city mothers contains less calcium than that of the well-nourished rural mothers, but the deficiency is slight and there is always left ample to provide for the child's requirements.

Growth.—It has already been pointed out that one of the salient features of the rachitic process is hyperplasia of the cartilage, and that it tends to affect those bones which are in process of the most active development. It is a fundamental truth that without growth rickets cannot result. It is for this reason that the disease not only occurs most frequently during the periods of most rapid growth (infancy), but also that the localisation of the mischief varies at different ages. In early infancy the most rapidly-growing cranial bones suffer most (craniotabes). In later infancy it is the bones of the trunk and limbs, and in later childhood it is those of the limbs which are chiefly affected. In the younger child, too, because there is active growth not only in length but also in thickness of the bones, the mischief is apparent subperiosteally as well as at the epiphyseal ends (Fig. 89), whereas in later childhood, because growth is chiefly in length, the changes are almost if not entirely limited to the epiphyses (Fig. 95). It is also because growth is essential for the development of rickets that the most rapidly-growing infants are the most susceptible, that marantic infants seldom present evidences of the disease, and that starvation by arresting growth causes healing in the disease experimentally induced in rats. When, however, the marantic infant recovers, and especially if growth is unduly rapid, as in cretins under treatment with thyroid,¹ rickets is apt to supervene.

Disease of the Ductless Glands.—By different observers different glands of internal secretion have been credited with playing an etiological rôle, either on the ground of the therapeutic effect of the administration of the gland, or as the result of the disease being supposed to develop after excision of the gland in animals. Lanz² in 1894 stated that good results were got in the treatment of the disease by thyroid gland extract, and Stoeltzner³ in 1900 recorded good results by treatment with suprarenal extract. In 1907 Basch⁴

¹ J. Thomson, *Brit. Med. Journ.*, 1896, ii., 618.

² O. Lanz, *Samml. Klin. Vortr.*, n. F., 1894, No. 98.

³ W. Stoeltzner, *Jahrb. f. Kinderheil.*, 1900, li., 73 and 199.

⁴ K. Basch, *Jahrb. f. Kinderheil.*, 1906, lxiv., 285.

believed that he had induced rickets in the dog through removal of the thymus gland, and Erdheim¹ (1911) after parathyroidectomy. Little credence can be placed on this work, as for the most part it was carried out prior to the introduction of the accurate quantitative dietetic experiments and the demonstration that confinement itself in some way induced the disease.

Infective Theories.—In 1898 Monpurgo² first suggested a bacterial origin, in view of an epidemic of a disease simulating rickets and osteomalacia in a colony of white rats. He isolated a diplococcus, inoculated other animals, and found that rickets developed; from the inoculated animals, however, he did not recover the organism. Tovane and Forte,³ Moussou,⁴ and Koch⁵ have also been successful in inducing rickets in fowls and dogs by inoculation with bacteria. The criticism raised against the excision of gland experiments, that neither the conditions of housing nor of diet were properly controlled, also applies to these experiments. In 1925, however, Kawamura and Kasama⁶ recorded success in inducing rickets in the rabbit by feeding with an animal parasite. It may be mentioned here that so far all attempts to cause rickets in the kitten have been unsuccessful. Such paradoxical findings as the above should make us critical of unreservedly applying results in the animal to conditions as met with in the human subject.

Environment.—According to Glisson rickets was more rife among the children of the rich and well-to-do, but in modern times the reverse is the case, as the disease almost exclusively affects the children of the poor. The deplorable hygienic conditions of the homes of the rich in those times is well known. It was the rich who lived in houses which were close and badly ventilated, whereas the poorer classes led a more or less open-air life. Nowadays the rich have learned to live in a more or less hygienic fashion. Not so, however, is it the case with the poor. Too often the poorer classes have to rest

¹ J. Erdheim, *Sitzungsberichte der kaiserlichen Akademie der Wissenschaften*, 1911, Band xcvi., Abt. 3.

² B. Monpurgo, *Beiträg z. path. Anat. u. z. all. Path.*, 1900, xxviii., 620.

³ A. Tovane and S. Forte, *La Pediatria.*, 1907, xv., 641.

⁴ G. Moussou, *Bulletin de la Société Centr. de méd. vet.*, 1903, xxi., 303.

⁵ J. Koch, *Zeit. f. Hyg. and Infekt. Krank.*, 1911, lxix., 436.

⁶ R. Kawamura and Y. Kasama, *Journ. Exp. Med.*, 1925, xlii., 793.

content with dwellings discarded by the more fortunate wealthier classes.

This class distinction in the incidence of rickets has naturally drawn special attention to environment as a possible etiological factor. When it was also observed that animals in confinement were subject to rickets, while living under natural conditions they were immune, it seemed fairly definite that somewhere in the complex of what environment speaks for the cause must be found. Kassowitz¹ suggested that it was the breathing of the noxious gases in the badly-ventilated houses which was the exciting factor. Hansemann² rested content by indicting what he called domestication, which for him was everything that characterised civilisation in contrast to Nature. Findlay,³ in the course of experiments in dogs on the blood changes in rickets, also became convinced that confinement was the cause, and concluded that lack of exercise was the important element in the complex which was responsible for the perversion of ossification. It was the apparent sufficiency of light and fresh air in the kennels, and the satisfactory diet which the animals were taking, as well as the effect of exercise in favouring calcium retention demonstrated by Kochmann and Petzsch,⁴ and the therapeutic effect of massage which forced him to this conclusion. Further experimental work done by Findlay and Paton⁵ seemed to confirm this view, and it is now generally admitted that this is one of the various factors which influence ossification.

Diet.—Glisson considered diet a contributory factor but, in contrast to modern opinion, he blamed a too rich and plentiful diet. He remarks that it may be for this reason that the "Disease doth more frequently invade the cradles of the rich than afflict poor men's children." Towards the end of the eighteenth century, at least to judge from the evidence of the famous Dr Buchan,⁶ this view of etiology was still prevalent. Buchan puts the causes as over-rich feeding, absence of breast-feeding, and, it is interesting to record, deficient exercise.

¹ M. Kassowitz, *Die Pathogenese der Rachitis*, Wien, 1885.

² D. von Hansemann, *Berlin. Klin. Woch.*, 1906, Nos. 20 and 21.

³ L. Findlay, *Brit. Med. Journ.*, 1908, ii., 13.

⁴ M. Kochmann and E. Petzsch, *Bioch. Zeit.*, 1911, xxxii., 10.

⁵ D. N. Paton, L. Findlay, and A. Watson, *Brit. Med. Journ.*, 1918, ii., 625.

⁶ W. Buchan, *Domestic Medicine*, Edinburgh, 1769, 483.

During the latter half of the nineteenth century almost every conceivable fault in the diet was credited with playing an etiological rôle, but those receiving most support were a deficiency of fat, a deficiency of protein, an excess of carbohydrate and a deficiency of lime. The excess of carbohydrate was said to induce acidosis which washed the lime out of the bones. It is self-evident how a deficiency of lime was supposed to act, but how the deficiency of fat or protein exerted their influence is not even hinted.

Although for long many clinicians believed that scurvy was an example of a diseased process induced by the absence from the diet of some specific substance, but of unknown nature, it was the work of Eijkman¹ in 1897 which gave a fresh outlook to the dietetic theory of the cause of rickets. Eijkman showed that fowl fed on polished rice developed neuritis, and that an alcoholic extract of the rice-polishings cured the condition. In 1906² Hopkins demonstrated that some vital matter in addition to the proximate principles and salts was necessary in the diet. The nature of this substance was quite unknown and was named by Hopkins the accessory food factor. It was at this time that Hopkins first suggested that rickets might be due to the absence of some such accessory food factor. By means of careful quantitative dietetic animal experiments, in which the various proximate principles and salts were arranged in varying but accurately known proportions, Hopkins definitely proved that there was such a factor (now called vitamin A) which influenced growth and which was associated with animal fat. Mellanby,³ a disciple of Hopkins, applied the same method of study to rickets and believed that he had demonstrated that animals did or did not develop rickets according to the absence or presence of this vitamin in the diet. It was, however, later shown that animal fat or cod-liver oil, which was specially rich in this vitamin A, could be so treated as to destroy the growth-promoting influence while leaving unaffected the antirachitic power.⁴ Hence it was

¹ C. Eijkman, *Virchow's Arch. f. path. Anat.*, 1897, cxlviii., 523.

² F. G. Hopkins, *Analyst*, 1906, xxxi., 395.

³ E. Mellanby, *Lancet*, 1919, i., 407.

⁴ F. G. Hopkins, *Biochem. Journ.*, 1920, xiv., 725 ; E. V. McCollum, N. Simmonds, J. E. Becker, and P. G. Shipley, *Journ. Biol. Chem.*, 1922, liii., 293.

necessary to speak of a special antirachitic factor or vitamin, originally called vitamin X but now called vitamin D.

When McCollum¹ of America resuscitated the use of the rat as an experimental animal in the study of rickets a great advance in our knowledge of dietary factors was made. Previously the dog had been almost wholly employed, but the rat has the advantage that it can be used on a much larger scale; it is cheap, easy to house, and it develops the disease quickly (in one-third of the time that it takes in the dog), so that much larger series of experiments can be conducted. About this time another innovation was made. Almost simultaneously Sherman and Pappenheimer,² and McCollum and his co-workers, found that a condition apparently identical with human rickets could be induced by feeding rats on a diet in which there was a deficiency of phosphorus. During the nineteenth century most experimentalists had essayed to produce rickets by feeding a diet poor in calcium. Although some workers, *e.g.*, Guerin (1838),³ thought they had been successful, it was ultimately realised, when Pommer in 1885 described the true histological picture of rickets, that the malady induced in this way was osteoporosis. The condition, however, which Sherman and others brought about in the rat was histologically true rickets, as evidenced by the erratic and hyperplastic growth of the cartilage and the production of osteoid tissue.

These American workers found that so long as the phosphorus and calcium in the diet were properly balanced, and adequate in amount, rickets would not develop although there was a complete absence of vitamin D. It was also shown that cod-liver oil in which the vitamin A had been destroyed, but which still retained vitamin D, cured or hindered the development of this variety of rickets. Thus as two factors, (*a*) deficiency of phosphorus, and (*b*) absence of the substance D, are necessary for the production of this type of rickets, it cannot strictly be referred to as an example of avitaminosis, in which the only fault is an absence from the diet of a specific factor. For this reason Hess and others object to calling the substance

¹ E. V. McCollum, N. Simmonds, H. T. Parsons, P. G. Shipley, and E. A. Park, *Journ. Biol. Chem.*, 1921, xlv., 333.

² H. C. Sherman and A. M. Pappenheimer, *Proc. Soc. Expt. Biol. and Med.*, 1921, xviii., 193.

³ J. Guerin, *Gazette Medicale de Paris*, 1838, vi., 332.

X or D a vitamin and prefer to speak of it as the antirachitic factor. If, indeed, it were a vitamin it is remarkable that it should be so rare in natural foods, as such substances are amply represented in a general dietary. The only natural food, milk, which contains all known vitamins, possesses a minimal and inadequate degree of the antirachitic power. In fact, it has been found that an excess of milk rather tends to the production of the disease. The only known food which possesses this antirachitic power to any extent is egg-yolk. Cod-liver oil, the medium *par excellence* of the antirachitic factor, cannot be considered in the true sense of the term a food. Hence, since it would not only appear that this disease in rats is not a true deficiency disease (avitaminosis), but that it is in part contributed to by a deficiency of phosphorus in the diet, it is difficult to appreciate the bearing which this variety of rickets can have to the disease as met with in the child. In the child a deficiency of phosphorus can be definitely excluded, and further, the natural diet of the infant at the period of life when the disease is most frequent is his mother's milk, which, as we have remarked, contains a minimal and inadequate amount of the substance X. The whole question, however, has been clarified and the probable cause of the relationship between diet, environment, and rickets revealed since the demonstration by Huldschinsky¹ that the ultra-violet rays cure human rickets, and since Hess² has shown that foods inert against both human and rat rickets can be rendered potent by exposure to these same rays. This aspect of the question will be discussed in the following section.

Sunlight.—In 1889 Huntly,³ a medical missionary in India, stated that absence of sunlight was the cause of rickets. He had just graduated and was imbued with the teaching of the day, as categorically expressed by Cheadle⁴ in 1888, that deficiency of the fat was the real etiological factor. Finding the diet of the native Indian very poor in fat, he was surprised at the absence of rickets among these people and suggested that absence of sunshine was the cause, since the great difference between life in India and Scotland (Huntly was educated at

¹ K. Huldschinsky, *Deut. Med. Wochenschr.*, 1919, No. 26.

² A. F. Hess and M. Weinstock, *Amer. Journ. Dis. Child.*, 1924, xxviii., 256.

³ W. Huntly, *Ajmere Rajputana Mission Press*, 1889.

⁴ W. B. Cheadle, *Brit. Med. Journ.*, 1888, ii., 1145.

Glasgow) was the relative abundance of sunlight. Many years later (1918) Hutchison,¹ another Glasgow graduate, found rickets in India rife among the better class Mohammedans (infants and children) who, on account of caste, practise *purdah* and so remain indoors during the daytime. In 1890 Palm² published the results of a study of the habits of the people in China, India, and elsewhere, and their climatic and sanitary condition with regard to the occurrence of rickets. Palm's findings were that rickets was most prevalent where sunshine was least and rare where sunlight was abundant. Unfortunately the work of Huntly and Palm received little attention, and it was not till 1919, when Huldschinsky demonstrated the curative effect of ultra-violet rays in rickets, that this factor obtained serious attention. Since then, however, an enormous amount of work on the subject has been done both in the laboratory and in the clinique. Not only have Huldschinsky's findings been completely substantiated, but it has been shown that radiation of rats in which rickets had been induced by the phosphorus-poor diet were as effectively protected and cured as by cod-liver oil. In 1924 Hess³ demonstrated that cotton-seed oil, normally quite inert against rickets, can be rendered rickets-curing by exposure to ultra-violet rays, and since then the same has been proved in connection with all foodstuffs. It has also been found possible to increase the potency of milk and yolk of egg, either by radiation of the foods themselves, or by radiation of the cow and hens. It has also been shown that the substance in the food which gives to it this antirachitic power is of the nature of a sterol (ergosterol) which, however, must be activated in some way by the ultra-violet rays of the sun or by those artificially produced.

From these various facts it is concluded that the protection of an individual or animal results from the ingestion of already radiated ergosterol, or by the production of the same in the body through exposure to the sun's rays. Hess⁴ has demonstrated that human skin, normally impotent against rickets in the rat, can be rendered potent by exposure to ultra-violet rays, and he suggests that the seat of production of radiated ergo-

¹ H. S. Hutchison and P. T. Patel, (a) *Glasg. Med. Journ.*, 1921, xcv., 241; (b) *Quart. Journ. Med.*, 1922, xv., 167.

² T. A. Palm, *The Practitioner*, 1890, xlv., 270.

³ A. F. Hess, *Amer. Jour. Dis. Child.*, 1924, xxviii., 517.

⁴ A. F. Hess and M. Weinstock, *Journ. Biol. Chem.*, 1925, lxiv., 181.

sterol is under the skin. The source of the factor in cod-liver oil, which of all articles possesses it in the highest degree, is supposed to be the plankton on which it feeds.

In summing up the lessons learned from all the recent work, it may be stated that we are still not certain of the real cause of rickets. It may be that it is due to the absence of the effect of the sun's rays, but this is mere deduction. We have seen that in rats rickets is caused by an absence of sunlight and an error in the salt content of the diet. The latter factor is certainly not at play in the child. We have also seen that in the rabbit the disease can be induced by the infection with a parasite. It is therefore possible that in the human subject some other factor or combination of factors is responsible. Nevertheless, the discovery that in radiated or activated ergosterol we have a substance which influences profoundly ossification is of far-reaching significance. The wide distribution of ergosterol not only in the plant but also in the animal world, and the ease with which it can be activated by the sun's rays, makes it very difficult to eliminate this factor from all experiments and more especially human tests. In the light of this factor we can understand the varying effect of the same article of food and of the same housing conditions. The secret is in the varying amount of direct or reflected sunlight which reaches either the plant or the interior of the home. The relative racial differences in susceptibility are also rendered comprehensible because the pigmentation of the skin offers a barrier to the penetration of the vital rays. And, finally, in this way it is also possible to explain the social distribution of the disease, its following in the wake of industrialism, and the benefit of everything which precludes confinement.

Prognosis.—Although rickets itself is not a fatal malady its presence may prove a dangerous complication in cases of respiratory disease. This is partly due to the way in which the softened chest tends to collapse and partly to the weakness of the muscles. If there is no such complication no case, however, is too severe to recover. With efficient cod-liver oil and the ultra-violet rays we are in possession of specific remedies which will certainly arrest the process, even although the hygienic and dietetic conditions cannot be changed. Any stunting of growth which has resulted will almost certainly persist, but the deformities, at least of the long bones, tend to disappear with

time. The rachitic conformation of the cranium, the enlargement of the ends of the bones at the wrists and ankles, and the deformities of the pelvis, however, persist.

Care must be taken not to mistake bony deformities from osteogenesis imperfecta and other conditions in later childhood for the stigmata of rickets. The existence of Macewen's tibial spine (Fig. 93), although not always present, is definite evidence of a rachitic origin of the deformity.



FIG. 93.—Macewen's tibial spine (A) in genu valgum.

Treatment.—As a result of recent work the therapy, both prophylactic and curative, has been simplified and rendered more certain.

(a) *Prophylaxis.*—In order to render the development of rickets improbable the child should be fed in a nourishing fashion and preferably at the breast. After weaning, yolk of egg, green vegetables, and an ample supply of good milk should form the basis of the diet. But perhaps more important is the general hygiene of the house and the amount of time the child spends in the open air. Confinement to the house, even behind vita glass, is the most certain way in which to cause rickets.

Children taken out every day seldom develop the disease even in the city.

If, however, there should be any fear that the above measures will not provide sufficient protection on account of prematurity, unduly rapid growth, excessive pigmentation of the skin (children of southern climes or negroes), or little opportunities for exposure to the sun, then the administration of cod-liver oil ($\frac{1}{2}$ drachm to 1 drachm thrice daily) will be found effective. All varieties of cod-liver oil are, however, not equally efficacious. In fact, some brands are quite useless. The varieties which are too refined should be avoided, and the darker brands are better than the lighter brands. The special preparations as radiostol are no more effective than good cod-liver oil and are in addition more expensive. It seems equally unnecessary to procure radiated cow's milk.

It is quite unnecessary to expose the child to the ultra-violet rays, as this method of prophylaxis is not only more expensive but the dosage is more difficult to control than in the case of cod-liver oil.

(b) *Curative treatment.*—When the disease is active, which, as already indicated, can only be definitely determined by either metabolic studies or X-ray examination, the child must be specially guarded against chill, as pulmonary mischief is particularly severe and difficult to eradicate in the rachitic patient.

The diet and general hygiene of the child must be attended to and rectified where necessary. If the disease is specially florid it may be advisable to keep the child off its feet for a short time.

In addition to the above general measures one of the definitely known methods of supplying active ergosterol must be employed; this may be efficient cod-liver oil or one of the synthetic preparations (radiostol or adexolin) or by exposure to the ultra-violet rays from the sun or mercury vapour lamp. Each and all of these methods of therapy are quite efficacious, and there is little difference between them in the rate of cure. If anything, the ultra-violet rays produce the most rapid cure, but there is very little in it, and in view of the expense and danger of this method it is not the therapy of election. The optimum dosage of the rays is difficult to estimate and the fact that arterio-sclerosis has been induced experimentally in animals renders its use not without danger.

Cod-liver oil, which is known to be effective, may be given in doses from one drachm to two drachms thrice daily. Within one week of the commencement of this treatment metabolic studies show a calcium and phosphorus utilisation which had been much below normal to increase to twice the normal, and within three weeks definite radiographic signs of healing are apparent. The bones are usually completely healed by the end of eight weeks (Figs. 89 and 90).

At one time oil of phosphorus was a recognised remedy. This is a one per cent. solution of phosphorus in olive oil and the dose is one to three minims thrice daily. It is doubtful if *per se* it has any effect on ossification, but it intensifies the action of the cod oil.

Fractures and deformities of the limbs from old healed mischief require the usual surgical and orthopædic measures.

Late Rickets.

As previously mentioned, rickets may relapse or appear for the first time in later childhood. When this happens after four years of age it is spoken of as *late rickets*, *rachitis tarda*, or *juvenile rickets*. Care must be taken not to mistake for a recurrence of the mischief merely the persistence of deformities which, on account of the fact that the older child tends to become sparer, may be rendered more noticeable. X-ray examination or metabolic studies can alone make the diagnosis certain. There is no doubt, however, that typical active rickets may develop during school age or adolescence, but it is not a frequent occurrence, at least in its most marked forms.

There seems no reason why this variety should be considered different from infantile rickets, as histologically and metabolically the same changes are observed, and since it reacts to the same therapeutic measures—cod-liver oil and ultra-violet rays. The only difference exists in the different distribution of the lesion, but this is no doubt due to the foci of most active growth being differently situated at this later age than during infancy.

Symptoms.—A child previously well complains of pains in the legs and back, becomes slow in his movements, and develops knock-knee or bow-legs. Because the most active growth at this older age occurs at the epiphyses it is there that the mischief may be alone apparent, or at least most marked.

Epiphyseal enlargement is often very evident, as also beading of the ribs. Bending of the bones is the next most frequent sign (Figs. 94 and 95).

Etiology.—It is usually considered that a dietetic factor is the more important in this variety of the disease, chiefly because

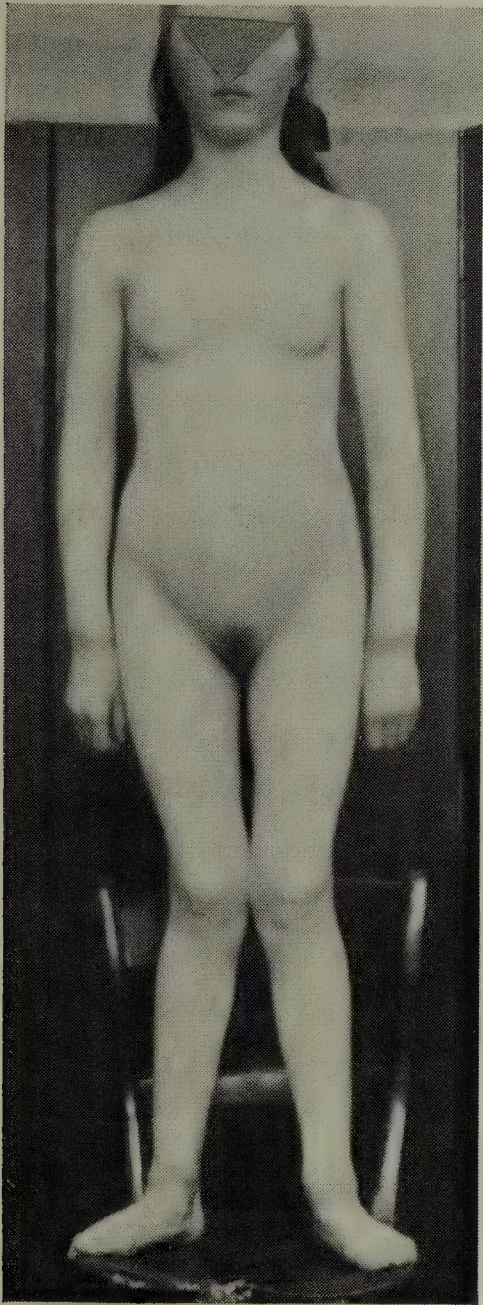


FIG. 94.—Late Rickets in a girl aged $14\frac{3}{4}$ years. Note double genu valgum and thickening of epiphyses at wrists and ankles.

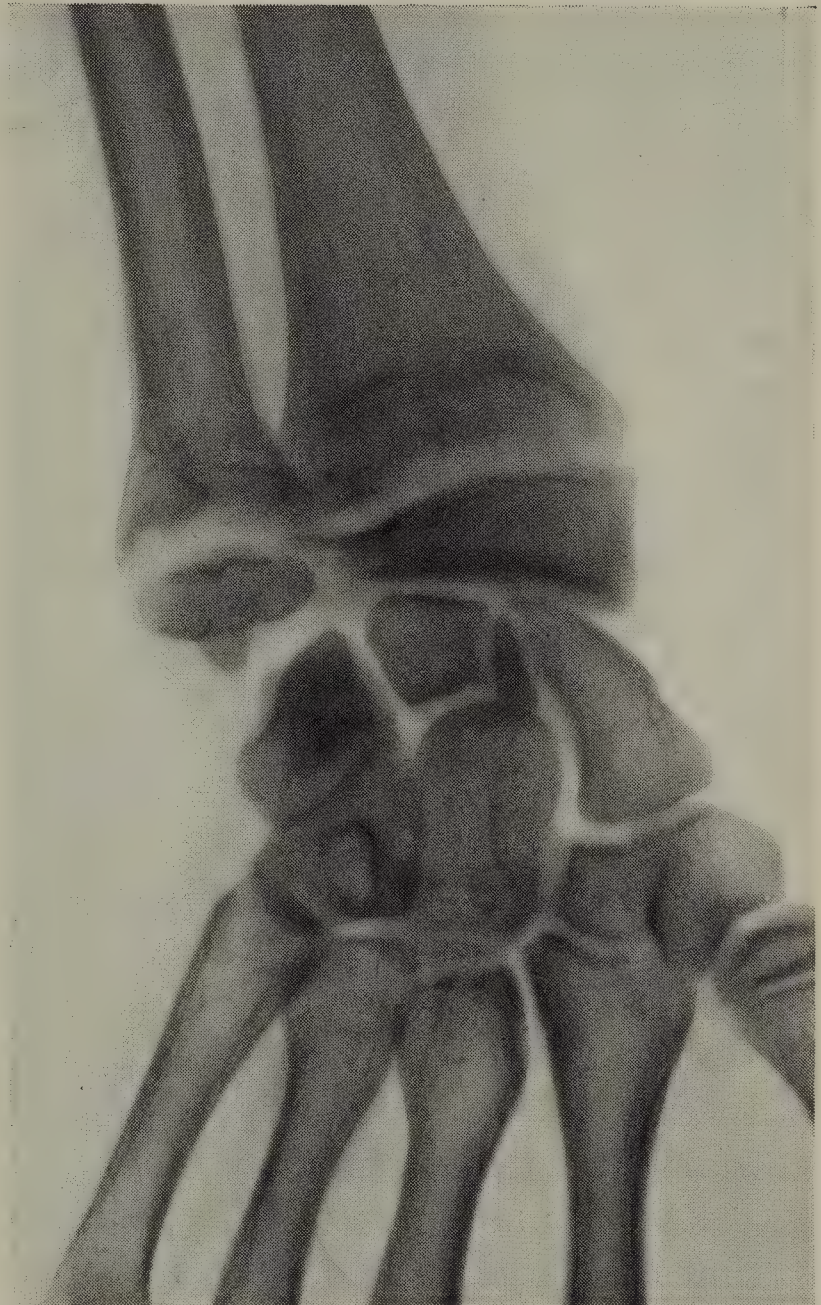


FIG. 95.—Skiagram of wrist in late Rickets (same case as Fig. 94). Note defective calcification in epiphyseal zone.

the disease was epidemic in Central Europe after the Great War. The exact fault in the diet, however, is never stated. In our experience the disease has always occurred in children who have been much confined to the house, just as was the case with the infantile variety of the disease. It may here be recalled that Hutchison found late rickets common in Moham-

medan girls practising *purdah*, although their diet was rich in animal fat (p. 274).

The treatment of late rickets is identical with that of ordinary rickets, viz., efficient cod-liver oil or one of the synthetic preparations containing radiated ergosterol, and exposure to the ultra-violet rays.

Rickets in Coeliac Disease.

Rickets with the radiological characters of rachitis tarda may also be met with in association with coeliac disease, and here also the question is discussed whether the association is a coincidence or whether it is a special type of the disease, having a special etiology and a special anatomy. Unfortunately no histological evidence is available to permit of a definite answer, but in view of the radiological evidence, and by analogy with rickets in renal fibrosis, we see no reason to look upon this type of rickets as a disease *sui generis*. We might here remark that the only undoubted example of lenticular degeneration observed by us during adolescence presented a severe degree of late rickets.

Very different are the incidence rates of the occurrence of rickets in coeliac disease as observed by different authorities. According to Parsons¹ it would appear to be not infrequent, whereas other observers, *e.g.*, Hess,² believe that it is a rare occurrence. In our experience the association is distinctly rare as we have only observed it once in twelve cases (Fig. 96). In this child the rachitic manifestations set in after recovery from the intestinal malady.

A certain degree of osteoporosis, however, is the rule and at times is very marked. This is not to be wondered at in view of the metabolic changes in this disease. In coeliac disease not only is there poor absorption of fat but also of protein and carbohydrate and of calcium and phosphorus as well. So far as these mineral substances are concerned, there is no difference between the metabolism in rickets and coeliac disease. In both there is a very diminished retention of lime and phosphorus.³ This in itself we know does not lead to rickets but to osteoporosis, which, however, probably predisposes to rickets if

¹ L. G. Parsons, *Arch. Dis. Child.*, 1927, ii., 198.

² A. F. Hess, *Rickets, Osteomalacia and Tetany*, 1929, p. 313.

³ O. MacRae and N. Morris, *Arch. Dis. Child.*, 1931, vi., 75.

the other necessary conditions are present. That osteoporosis rather than rickets is the rule at the height of cœliac disease, and that the latter only develops after recovery, is because one of the essential conditions, viz., growth, is arrested during the course of the disease and is only resumed on recovery. But



FIG. 96.—Skiagram of Rickets with fracture of ulna during convalescence from cœliac disease. (Boy aged 8 years.)

even a diminution in calcium and phosphorus in the presence of growing bone will not necessarily induce rickets. We know, however, that in the calcium-poor premature infant an excessive amount of vitamin X or D is necessary if ossification is to proceed normally, and it may be that the cœliac patient after recovery also requires an excessive amount of this same protective vitamin, and that rickets develops in cœliac disease because there is in some way an insufficient amount of the

vitamin available. In any case, ultra-violet rays and cod-liver oil do bring about healing, although it is admittedly slow, and Morris and MacRae have shown that cod-liver oil even during the acuter phases of the disease causes a definite increase in the retention of calcium.

Renal Rickets.

The presence of rickets in children the subject of chronic interstitial nephritis had been described in 1883 by Lucas,¹ and in 1890 by Goodhart,² but it is only since 1911, when Morley Fletcher³ recorded a case and suggested that the renal disease was the etiological factor, that the association has been considered cause and effect.

Although it is impossible to express any opinion regarding the age of onset of the nephritic element, the rachitic changes seldom make their appearance before the sixth year. Some observers have suggested that the nephritis may be congenital in origin, and the combination of rickets and nephritis has been observed by Paterson⁴ in a child aged twenty-two months.

The first changes observed are usually absence of growth with polydipsia and polyuria, and later genu valgum, or other rachitic deformities which in some cases assume a severe degree. The dwarfing is also often extreme, and is responsible for the earlier synonyms, *renal infantilism* or *dwarfism*. X-ray examination of the bones may reveal typical rachitic changes, or merely an osteoporotic condition. The urine has a low specific gravity and may or may not contain a small quantity of albumin, but the pigment test when applied has invariably demonstrated marked renal inefficiency. Cardio-vascular changes (cardiac hypertrophy and a raised blood pressure) may be present.

The duration of the illness may extend to many years, but ultimately death results from uræmia. Therapeutic measures are usually of no avail.

The chief interest in this disease is the pathogenesis and its relationship to infantile or ordinary late rickets. The bulk of

¹ C. Lucas, *Lancet*, 1883, i., 993.

² J. F. Goodhart, *Keating's Cyclopædia of Dis. of Child.*, London, 1890, iii., 555.

³ H. M. Fletcher, *Proc. Roy. Soc. Med.* (Sect. Child. Dis.), 1911, iv., 95.

⁴ D. Paterson, *Brit. Journ. Child. Dis.*, 1921, xviii., 186.

modern opinion ascribes it to phosphorus retention consequent on the nephritis, and thus an example of rickets of endogenous origin. Following on the retention of phosphorus there results a relative calcium deficiency. For this reason renal rickets is considered analogous to the low-calcium form of the disease induced in rats by low-calcium feeding. The blood findings—a normal or slightly lowered calcium and a high phosphorus—are taken as substantiation of this point of view. It will be remembered that in ordinary infantile rickets the calcium content of the blood is normal, whereas the phosphorus content is definitely diminished.

It is very doubtful, however, if the blood findings justify any such far-reaching conclusions. Ford,¹ in a recent study of the metabolism of calcium and phosphorus in nephritis, has shown that there is not a retention of phosphorus, but an almost absolutely parallel diminished retention of both calcium and phosphorus, the identical fundamental metabolic change observed in infantile rickets (p. 262). We would also point out that the same is true in tetany *per se*, or in tetany complicating rickets, and, as Morris and MacRae have recently shown, in coeliac disease as well. In all these conditions there is the same diminished retention of calcium and phosphorus, and yet the absolute and relative quantities of calcium and phosphorus in the blood are very varied. In rickets the calcium is normal and the phosphorus subnormal, in tetany calcium subnormal and phosphorus normal, in nephritis calcium normal or subnormal and phosphorus much increased, and in coeliac disease calcium and phosphorus both tend to be subnormal. In short, the blood picture cannot be taken as an index of the metabolism, or as proof of increased or diminished retention of any element. It would, for example, be ridiculous to conclude that the cause of the rachitic process had become different because on the development of tetany the blood picture had changed from one with a normal calcium and low phosphorus to one with a low calcium and high phosphorus, and there seems as little justification for considering, at least on the strength of the blood examination alone, another etiological factor in rickets complicating nephritis.

In nephritis, without rickets, the excretion by the urine of both calcium and phosphorus is diminished, but there is an

¹ F. J. Ford, *Arch. Dis. Child.*, 1931, vi., 209.

excessive excretion by way of the fæces, and it is this which is responsible for the diminished retention. Hence, not only in rickets but also in nephritis, the fundamental metabolic change is a diminished retention of calcium and phosphorus. The marvel rather is that rickets does not more commonly occur in nephritis considering the duration of the mischief and the slowness with which conditions return to normal on the recovery of the renal lesion. Nephritis must gradually bring about an impoverishment of calcium phosphate in the bones, which, as we have seen, is not infrequently reflected in the osteoporosis. When we remember that the premature infant is specially subject to rickets because it starts life with an inadequate quantity of lime, it is justifiable to conclude that the nephritic patient, and the coeliac patient as well, who are likewise starved of these elements, should also be equally susceptible to the disease. It is, of course, not merely impoverishment of calcium and phosphorus which brings about rickets, but some other factor of which we are still ignorant. Is it possible that here again the ultra-violet rays are the main deciding factor? The chronic invalidism accompanying the severer manifestations of the disease necessitate much confinement to the house, and thus an absence of this ossifying regulating factor. There is, however, another point which must not be lost sight of in this connection. We have seen that for the development of rickets growth is essential, but in the chronic nephritic, dwarfism or absence of growth is one of the most prominent features.

On histological grounds, too, there is little evidence for considering renal rickets different from ordinary rickets. Dr Haswell Wilson has had the opportunity of examining some of Parsons' cases, and has described the presence of osteoid tissue which we take as the final proof of the rachitic process.

The poor response to specific treatment in renal rickets is not to be wondered at. Some authors, *e.g.*, Parsons, believe they have seen recovery, but the majority of writers record no benefit from any therapy. Unfortunately for a complete answering of this question sufficient data are not available. There are no records of metabolic studies in renal rickets, and we are ignorant of the effect of cod-liver oil and the ultra-violet rays on the calcium and phosphorus metabolism in nephritis, such as is available in the case of coeliac disease.

CHAPTER XII

THE CLINICAL EXAMINATION OF THE ABDOMEN AND SOME DERANGEMENTS OF THE DIGESTIVE SYSTEM IN OLDER CHILDREN

Physical Examination of the Abdomen

Inspection.—In the early years of life the abdomen is relatively large. This is accounted for to some extent by the size of the liver, and it is rendered more noticeable by the comparative narrowness of the child's thorax.

Abnormal enlargement of the abdomen is common. It may be due to increase in size of one or more of the organs, to ascites, or to tumour. Much the commonest cause, however, is flatulent distension of the bowel or stomach.

On inspecting a distended abdomen we should note whether the enlargement is uniform or confined to one region only, such as that of the stomach, colon, or small intestine. In cases of chronic distension it is also well to be on the outlook for *abdominal patterns*.¹ These are formed by the occasional standing-out in rigid spasm of the distended small intestine, colon, or stomach, which is often accompanied by vigorous peristaltic waves. This is a very important symptom, because it generally indicates not only that the viscus in question is dilated, but that its muscular wall is hypertrophied as the result of long-continued overaction due to interference with the free passage onward of its contents. In older children in this country, far the commonest cause of this obstruction is abdominal tuberculosis, which acts either by producing narrowing of the bowel from the cicatrization of ulcers, or its compression by peritoneal adhesions (Fig. 336, p. 966). Such obstruction is commonest in the ileo-cæcal region; and evidence of its presence calls for early surgical treatment—either excision of the narrowed portion of gut or, more usually, the formation

¹ John Wyllie, "The Diagnostic Value of Patterns of Abdominal Tumidity," *Edin. Hosp. Rep.*, 1894, ii., 19.

of an anastomosis between the portions of bowel above and below it. Visible intestinal peristalsis is also a characteristic phenomenon in cases of congenital hypertrophy of the colon (Fig. 116, p. 357), but in very young infants it more commonly indicates the presence of congenital atresia or stenosis of some part of the intestine (Fig. 115, p. 356).

When well-marked visible peristalsis of a distended stomach is observed in an infant between ten days and four months old, this almost always signifies the presence of congenital hypertrophy of the pylorus. To be characteristic of this condition, however, *the peristalsis must be very distinct and forcible*; for in many wasted babies and older children with no obstruction the normal movements of the stomach and bowel are easily visible owing to the thinness and tonelessness of the abdominal wall.

On inspection of the abdomen we must also notice if there is any abnormal dilatation of the superficial veins, if the umbilicus is protruding, and if there is any redness of the skin round about it, as is common when an abscess is forming from abdominal tuberculosis. In cases of wasting in young children, the skin of the abdominal wall often presents little colourless elevations scattered over its surface. These look like raised nodules; but on touching them they are found to be quite soft; they are caused by dilated lymph spaces. In severe and protracted cases of diarrhœa, pyelonephritis, or tuberculosis, we sometimes find an extensive eruption of small ecchymoses in the skin of the abdominal wall. These indicate a dangerous though not necessarily fatal degree of weakness.

Retraction and hollowing-out of the abdomen are significant indications of intracranial disease (Fig. 236, p. 780) and diaphragmatic hernia; they also occur to some extent in uncompensated acidosis.

Palpation.—There are few methods of clinical examination so important in childhood as the palpation of the abdomen; and, unless tenderness is present, there is none that is less objected to by the patient, provided it is carried out tactfully *with warm hands*. It is a part of the investigation of the case which must never be omitted, even although no abdominal symptoms are complained of.

In many cases it is advisable to begin the palpation by slipping the hand under the clothes or bed-clothes, before the child has been disturbed by any other examination. While

this is being done, his attention should be turned as much as possible in other directions. If we succeed in this, it will usually be easy to ascertain whether pressure gives rise to any real pain, which is always a valuable fact. If, however, by roughness or cold hands his attention is drawn too much to what we are doing, we shall get little information and the chances of error are considerable.

It is always important to judge of the presence or absence of tenderness by observing whether the child winces on pressure being applied, and not by asking him if it hurts.

If pressure on any part causes pain, we have next to investigate whether the pain is elicited by superficial or only by deep pressure. If the former is the case, we should ascertain the exact site and superficial area of its distribution; and we must also endeavour by gently pinching up the skin between the forefinger and thumb, to discover to what extent the tenderness is situated in the skin, in the subcutaneous tissue, and in the muscles. This line of inquiry does not, indeed, give us important information so often in children as it does in adults, because many of the abdominal lesions (such as gastric ulcer, gall-stones, and urinary calculi) which give rise in later life to such a *viscero-sensory reflex*,¹ only rarely occur in childhood. It may, however, occasionally throw interesting and valuable light on the diagnosis in such conditions as acute pyelitis, tuberculous bowel lesions, subphrenic abscess, appendicitis, and other localised diseases in the abdominal cavity. Areas of extreme tenderness are also sometimes present on one or other side of the abdomen in cases of basal pneumonia or diaphragmatic pleurisy. In severe cases of general peritonitis the sensation may be dulled to such an extent that no pain is caused by pressure.

The *viscero-motor reflex*,² that is to say, the persistent involuntary contraction of muscle in the abdominal wall due to a nervous stimulus originating in a viscus, is often met with in children, and may be very useful in drawing attention to disease of the abdominal organs. It must be distinguished from the passing, involuntary contraction of the abdominal wall which accompanies rough handling or tickling, and also from the voluntary stiffening of the abdomen which occurs when the

¹ James Mackenzie, *Symptoms and their Interpretation*, 1906, 59.

² *Ibid.*, 67.

child is in a state of apprehension. The visceromotor reflex is generally present to a marked degree whenever there is pain or pressure arising from any of the structures in the abdomen, but it is often found also when there is none. Owing to the peculiar character of the innervation of the abdominal muscles, the contraction usually affects small segments only of the muscular tissue, and the hard contracted areas thus produced are frequently mistaken for tumours of the underlying organs.

The softness of the abdominal wall in children allows enlargement of any of the organs and the presence of tumours, inflammatory swellings, and abscesses to be more easily felt in them than in adults. For the satisfactory palpation of the abdomen, however, in difficult cases, the administration of an anæsthetic is often necessary in order to allow a thorough combined abdominal and rectal examination to be made.

Percussion.—Although not nearly so important in the examination of children as palpation, percussion is also useful in determining the state of the stomach and bowel and the relation of the latter to tumours, and in confirming the results of palpation. It is also of great value in ascertaining the presence of free fluid in the peritoneal cavity.

The Liver.—The lower edge of the liver is usually best made out by palpation. It reaches farther down in children than in adults, and often in health is at least half an inch below the costal margin in the right mammillary line. This is due, partly to the liver being relatively large, and partly to the ribs lying more horizontally than in later life, and consequently leaving more of it uncovered (Sahli).

Diminution in the size of the liver-dullness is rare in childhood. It occurs in acute liver atrophy, in the later stages of cirrhosis, and to a less degree in coeliac disease. Disappearance of the dullness is a valuable sign of the presence of gas in the peritoneum which occurs when gastric or intestinal ulcers have perforated.

Hepatic enlargement is common and may be due to many causes, far the commonest of which is fatty accumulation. It is well to remember that a large fatty liver, though flabby after death, has often a hard, sharp edge during life. A large liver is also found in passive congestion from heart-failure or from mediastino-pericarditis, in tuberculosis, lardaceous disease, catarrhal jaundice, the early stage of atrophic

cirrhosis, Hanot's hypertrophic cirrhosis, hepatic neoplasms, and Gaucher's disease.

The Spleen.—Like the liver, the spleen is best investigated in children by palpation. To examine it, one should stand on the child's right side and, laying the right hand with the first two fingers over the left hypochondrium, press gently inwards and upwards. When this is done, its edge will be felt as a rounded, freely movable body if the organ is enlarged, and also in some cases in which it is normal in size. When beginning to palpate, it is generally well to make sure of the position of the cartilages of the lower ribs, as they are apt to be mistaken for the border of an enlarged spleen. If the child allows free palpation and the spleen is not felt at all, one may generally be sure that it is not enlarged. A large tuberculous spleen may occasionally, however, be beyond the reach of the fingers owing to its being adherent to the under surface of the diaphragm.

The common causes of enlargement of the spleen are acute infections (enteric fever and septicæmia), chronic infections as syphilis, tuberculosis, lymphadenoma (Hodgkin's disease), blood diseases (splenic anæmia, acholuric jaundice, and leukæmia), Gaucher's disease, and lardaceous disease. The spleen is also enlarged in cirrhosis of the liver and in catarrhal jaundice, and in thrombosis of the splenic veins. In children who have been resident in the tropics malaria is a possible etiological factor, and in those who have resided on the shores of the Mediterranean and in India an infection by Leishman-Donovan bodies may be the cause of an enlarged spleen (p. 474).

Rickets is not a cause of splenomegaly, but from deformity of the chest the organ may be displaced downwards and appear as if enlarged.

The Mesenteric Glands.—Enlargement of the mesenteric glands is due in rare instances to tumour formation; and, occasionally, a slight palpable enlargement may be found also in non-tuberculous ulceration of the bowel or non-tuberculous peritonitis. In the enormous majority of cases in this country, however, the enlargement is due to tuberculosis, and it is often associated with the formation of palpable caseous masses in the omentum and elsewhere. Tuberculous glands may be present in any part of the abdomen, but they should be examined for specially in the ileo-cæcal region, behind the

umbilicus, below the liver, and in front of the vertebral column. In some cases they are so deeply situated that they can only be felt when they are very large and hard; and sometimes they can only be discovered under an anæsthetic by rectal or combined rectal and abdominal examination.

Rectal Exploration.—In abdominal conditions a rectal examination should never be omitted. A simple examination with one finger may be sufficient, as in searching for a rectal polypus; but a bimanual exploration should be made in most forms of abdominal and pelvic disease. It is especially important in cases of obstinate constipation, in spinal disease with possible abscess formation, in those of tumour and abdominal tuberculosis, and in the “acute abdomen” for the discovery of a pelvic appendix abscess. The patient, having been anæsthetised, is laid on his back with the thighs fully flexed on the abdomen and a pillow under the pelvis. With the right forefinger in the rectum and the left hand over the front of the abdomen, the right side of the pelvis and abdominal cavity, as far as can be reached, may now be explored satisfactorily. The physician then stands on the child’s left side and examines with the left forefinger in the rectum and the right hand palpating in front. In this way, besides feeling enlarged glands or other swellings, he may be able to discover any thickening or matting of the intestine that has been left by former inflammatory attacks.

Hernia.—*Umbilical hernia* is very common in infants. It is free from danger, and if treated by infolding it, with the abdominal wall in the middle line, and keeping it in this position by broad strips of adhesive plaster, it usually recovers completely in a few months. Plastic operations are sometimes performed, but it should be remembered that the condition shows a strong tendency to disappear, as is evidenced by the rarity with which it is encountered in the adult unless from definite disease.

Inguinal hernia is far commoner in boys than in girls (nine to one); in both sexes it is much more frequently found on the right than on the left side, but it is often bilateral. In slight cases, if the child is carefully nursed and fed so that crying and straining are reduced to a minimum, the hernia often disappears—with or without the use of a truss. The recovery under these circumstances, however, is not always

complete; and a small canal is apt to be left, through which the hernia may return in after-life as the result of a sudden strain. To prevent the possibility of this occurring and also to save the child from the great discomfort of having to wear a truss, it is practically always advisable that cases of inguinal hernia should be operated on in infancy. The operation is now almost entirely devoid of danger and is practically always successful. It is well to have it done before teething begins.¹

Femoral hernia very rarely occurs in childhood.

Rectal Polypi.—When an apparently healthy child repeatedly passes a small amount of pure blood immediately after defæcation, the presence of a rectal polypus should always be suspected.

Rectal polypi are of the nature of adenomata, and they are most frequently situated on the anterior wall, an inch or more from the anal orifice. They are generally soft in consistence, rounded in shape, and pedunculated; many of them reach the size of a bean or small cherry. Occasionally they are seen protruding from the anus after defæcation. The polypus is usually easily felt on rectal examination, but sometimes, if very soft, it may be difficult to make sure of it.

The treatment consists in seizing the tumour with dressing forceps or with a wire snare, and twisting it off. The cure is immediate and permanent.

Signs of Imperfect or Deranged Digestion.

Disorders of Appetite.

Diminished Appetite (*Anorexia*).—Temporary disinclination for food is shown by most children during the onset of acute disease. Accompanying as it does a lessened power of digestion, it is to be looked upon as natural and beneficial. Continued refusal of food, however, is a different matter and always calls for investigation and treatment, especially if it has lasted long and is obviously interfering with the child's nutrition. In delicate children with tuberculous lesions, a small appetite is often a serious evil, because it interferes greatly with the fattening process which is so much to be desired in them.

Many parents are under an erroneous impression that the

¹ H. J. Stiles, "On the Operative Treatment of Hernia in Infants," *Brit. Med. Journ.*, 1904, ii., 813.

child's appetite is not vigorous enough and complain that he does not take sufficient food. These parental impressions can be gauged by a study of the fæcal output and the state of nutrition of the child. Sometimes, however, the inclination for food may be interfered with and for several reasons.

1. In a few cases it is caused by a painful condition in the mouth or throat, such as stomatitis, dental caries, or pharyngitis, and will pass off when these are attended to.

2. Confinement to the house and lack of exercise and interest are even commoner causes in children than in adults; and, by whatever the loss of appetite is caused, a change of air and surroundings will often work wonders.

3. In many cases the disinclination to eat is due to anæmia or debility. In the former, a grain or two of citrate of iron and quinine, and in the latter, dilute hydrochloric acid (1 to 3 minims) or bicarbonate of soda (3 to 5 gr.) with tincture of nux vomica (2 to 4 minims) thrice daily, well diluted, before meals, will generally be found useful.

4. The appetite is often discouraged by the child being given food too frequently because he takes so little each time. When this is so, the stomach has no time to empty itself before it is called upon to deal with a fresh supply of food; and a lengthening of the intervals causes a marked improvement.

5. In babies a too large proportion of cream in the milk is a common cause of loss of appetite; and the same may be said of the administration of cod-liver oil in older children.

6. Distaste for food commonly accompanies constipation, and it tends to disappear when the bowels begin to act regularly and freely.

7. Loss of appetite is one of the common symptoms of chronic intestinal indigestion, which is generally greatly improved by suitable changes in the diet (p. 301). In these cases, also, the internal use of ichthyol (1 to 2 gr. t.i.d.) has often a strikingly beneficial effect.

8. In many, probably in most, cases of refusal of food in young children the defect lies mainly, if not altogether, in the child's mental condition, and the only efficient treatment consists in a careful and judicious training of his habits of life, such as he has probably not had before.¹

¹ See an interesting account of this condition in *The Nervous Child*, by H. C. Cameron, London, 1919, 50.

We find that in the past he has been unwisely accustomed to expect almost everything to be done for him, but that he has early learned that one thing that he can always do without trouble to himself is to refuse his food. He has found that his doing so gives rise to a pleasant sense of importance and power on his part, and a gratifying amount of disturbance on that of his mother and nurse. This habit of refusing food increases greatly at times when he is out of sorts or out of temper, and every meal becomes a struggle between him and his seniors. Their evident anxiety on the subject magnifies its importance in his eyes; and the various devices they employ to make him eat—such as telling him stories during meals, persuading him to take a spoonful from each member of the household, etc., etc., have only a very temporary effect. The act of eating gets less and less spontaneous, and ultimately he becomes convinced that he cannot possibly swallow the food, and each attempt to make him do so ends in vomiting. Whenever a child shows a tendency to this sort of conduct, trouble should be taken to increase his initiative; and his natural tendency to imitate his elders should be fostered in every way. He should be made to do everything possible for himself, and be waited on only when it is quite necessary to do so. He should be made to feel that the only one really interested in his appetite is himself. It is in children who are used to being fed, and not in those who have been encouraged to show their cleverness by feeding themselves like grown-up people, that such difficulties arise.

The attitude of the mother and nurse to the subject is the important matter. As already indicated, anxiety and fussiness on their part are fatal, while a confident, unemotional, off-hand manner will often work wonders. It is also important that, in the intervals between meals, the child's refusal of food should never be alluded to in his presence. Any mention of it constitutes a pernicious suggestion which is sure to favour its recurrence. The least hint given that he is not expected to eat well makes it more difficult for him to acknowledge his past error by giving up his bad habit. When he shows the slightest improvement, however, it may do good to emphasise this by showing him that it is taken for granted now that he has no longer the difficulty he used to have and that all concern about the matter is at an end.

Cases of this kind may take a long time and give a great

deal of trouble; but, if those in charge are sensible, and have the nature of the trouble clearly explained to them, they will often show a natural skill in "mother-craft" that soon puts things right.

Increased Appetite (*Bulimia*).—An excessive desire for food is sometimes a sign of indigestion; often it is merely the result of a bad habit; it is common in obese children, but in diabetes in childhood it is not a prominent symptom.

Depraved Appetite (*Pica*).—In some otherwise normal children—as well as in many who are neurotic or mentally deficient—there is at times an unnatural craving for earth, coals, lime, or other things unfit for food. This is apt to be aggravated when anæmia, intestinal indigestion, or worms are present. (See *Pica*, p. 702.)

Gastric Flatulence.—In older children, as in infants, an excess of air in the stomach is always due to air-swallowing and not to fermentation of the food. When the wind is swallowed, it rarely gives rise to much discomfort so long as the child is going about, as it is usually brought up again. When children, however, are kept lying on their back for any reason, as for spinal caries, the abdominal distension caused by air-swallowing may become distressing, and in weakly ones even dangerous. In these circumstances it may be necessary to raise the patient from time to time to a vertical position to allow spontaneous eructation to take place.

Intestinal Flatulence.—When an indigestible excess of starch, milk-curd, vegetables, or fruit is taken, the normal amount of wind in the bowel is greatly increased. By lessening these elements in the diet the formation of excessive flatulence can usually be stopped at once.

Intestinal Colic.—Ordinary colic is due to violent peristaltic contractions of the small intestine, and the pain is always referred to the middle line in the region of the umbilicus. It is set up, in older children, by the same causes as in infancy (p. 149), is recognised by the same characters, and relieved by the same treatment.

Vomiting and Diarrhoea.—The causes of vomiting and diarrhoea have already been considered (pp. 148 and 151).

Constipation.—The causes and treatment of constipation in infancy have also been already described (p. 152). A few points may now be dealt with regarding the condition as it appears in

older children. When a child is brought to us with this complaint, it is well to begin by inquiring whether it is of long standing or only a recent development and, if recent, as to the circumstances under which it has arisen.

If the constipation is of long standing it may, in rare instances, be due to anal narrowing, megalocolon, or some other anatomical peculiarity. In the large majority of cases, however, it arises from defective training, from habitual injudicious diet, or from general bodily and mental atony.

If it has lasted only a short time, we must inquire as to recent bowel disorder (*e.g.* colitis), and not forget the possible presence of a rectal fissure (p. 137). This latter condition is not very rare, and it is important that, if present, it should not be overlooked, because of the special treatment it requires. In older children, especially in girls, severe constipation sometimes comes on as a hysterical symptom along with mental depression.

Habit.—The formation of a habit of having the bowels moved regularly is a most important point in the treatment of most cases of constipation; and our other methods of treatment should all be used mainly with the view of establishing such a habit. It is always desirable to see to it, not only that the child gets into the way of going to the closet regularly after breakfast, but also that his other morning duties are so arranged that he shall not be hurried unduly in the performance of this important one. The normal habit of having the bowels emptied regularly is often interfered with by the same mental attitude of “negativism” which causes refusal to take food (p. 292), and this has to be treated on the lines already indicated.

Diet.—Constipation in children, as in adults, may be due to the food given being too completely digestible. Quite as often, however, it is kept up by an excess of incompletely digestible substances such as oatmeal porridge, brown bread, raw fruit, and vegetables which are being given him with the idea that they are necessarily laxative. In many long-standing cases these things fail altogether to stimulate the bowels as they are expected to do, and then they set up chronic intestinal indigestion with large undigested motions which are difficult to get rid of. In such cases, a complete change of food—malted cereals, “malted milk,” fruit juice, jellies, meat, eggs, and soups—is often followed promptly by regular motions. A diet

containing too much white bread, farinaceous puddings, and milk is also, of course, objectionably constipating.

Mechanical Aids.—At the beginning of the treatment in a bad case, glycerine suppositories or injections, or small soap-and-water enemata, may be useful in helping to form a regular habit, but these measures should only be used temporarily, and for this purpose. If they are continued for long, the child gets into the way of trusting to the artificial stimulus they supply, and his constipated tendency remains.

Massage.—Along with the regulation of the diet, the use of skilled massage may be of great value in establishing regular ways. In hysterical cases, a course of Weir-Mitchell treatment has sometimes, in my experience, been most successful.

Medicines.—As already said, drugs are mainly to be used to initiate and foster regular habits. Cascara with malt extract or rhubarb (Appendix E, Form. 25), infusion of senna pods, other preparations of senna such as “Fig Syrup,” various fruit pastes which chemists prepare, and heavy magnesia or phosphate of soda (q. s. with all meals) are all sometimes useful. So also are “regulin” (agar-agar) and phenolphthalein; and a course of a purgative mineral water or of Carlsbad salts may sometimes prove beneficial. In all cases, the careful regulation of the dose necessary is important; and it should be gradually lessened as good habits become established.

Incontinence of Fæces.—Habitual involuntary passage of the motions may be due to paralysis of the sphincter in such conditions as Pott’s disease and spina bifida. It often comes on temporarily from mere weakness in children exhausted by diarrhoea and other debilitating conditions; and in nervous children when indisposed, as during the onset of an acute illness. In such cases, no local or special treatment is called for.

A different type of case sometimes occurs, apparently as the result of inco-ordination similar to that in enuresis. It is indeed often associated with that symptom, and is much more frequently diurnal than nocturnal. This kind of incontinence is often, though not always, seen in nervous anæmic children, and in those who are stupid and ill-regulated and show a degree of mental instability. The motions, though they may not be too numerous, are often unhealthy in character. This condition is common in the mentally deficient.

Treatment.—(1) If debility is present, means should be taken to improve the general health by tonics such as iron, arsenic, and strychnine, and by the administration of cold douches. (2) The digestion must be attended to, and if the motions are unhealthy or there are other reasons for suspecting the presence of intestinal indigestion, it will be well to limit the amount of starchy food taken and to stop uncooked fruit and the coarser vegetables for a time. (3) Ergot and belladonna are sometimes of use, as in incontinence of urine, and Still recommends a course of Dover's powder (1 to 3 gr.) thrice daily. (4) At the beginning of the treatment it sometimes helps in restoring the child's confidence in himself if the bowel is emptied every morning for a time by an enema. (5) Very frequently, the psychical part of the treatment is the most important. The bad habit is, for example, checked at once in many cases by the mental effect of removal to a hospital ward. Hensch recommended the subcutaneous injection of ergotin (1 gr.); he suspected, however, that the good effect of this was largely due to its painfulness.

CHAPTER XIII

SOME DISEASES OF THE DIGESTIVE SYSTEM

Chronic Intestinal Indigestion (*Mucous Disease*)

CHRONIC intestinal indigestion is the name which is now usually given to a very common form of dyspepsia, the main feature of which is an inability to digest and absorb with comfort an ordinary amount of certain common articles of diet—especially vegetable and starchy foods. It is present to some extent in a large proportion of delicate children. In slight cases the symptoms may be so trivial as to escape notice, the child being brought merely because he is not gaining weight or seems out of sorts. In severe cases, however, they may be distressing, and occasionally may simulate closely those of grave organic disease. From the point of view of general practice there are few maladies of early life more important, because it is so commonly met with, causes so much worry and anxiety, and yet is so amenable to proper treatment. It is found in all ranks of society, and is especially frequent and severe in the children of gouty and neurotic parents. It often appears between the third and fifth year, but is more common in older children, and it generally, even in severe cases, is recovered from long before adolescence.

Symptoms.—These may be divided into three groups: (1) there are indications that something is interfering with the child's growth and nutrition; (2) there are various signs that the digestive organs are out of sorts; and (3) there are derangements of the nervous and other systems, obviously due to auto-intoxication.

(1) The patient is usually thin and delicate-looking; he is often slightly built and looks young for his years. He is pale-faced in most cases, although his mucous membranes may show no anæmia. The skin is generally soft and normal. The limbs are slender and feeble, and all the muscles poorly

developed. The hands and feet are apt to be cold and flabby. Not infrequently there are symptoms of the presence of adenoids.

(2) On investigating the state of the alimentary tract, we find various more or less indefinite signs of derangement. The tongue may be fairly clean, but usually it is covered with a slight brownish fur and has a peculiar slimy appearance. In bad cases the breath may be offensive, but often this is not so; there is frequently a short dry cough—a “stomach cough.” The abdomen may be more or less distended, although nothing further can be made out on palpation. The bowels are generally constipated, the motions being pale and pasty, sometimes clay-coloured, and sometimes very offensive. Occasionally there is chronic diarrhœa with large foul-smelling stools, and lenteric attacks may occur. On examination of the motions, an abnormal amount of mucus is usually found; and, when this is not apparent on naked-eye examination, it may be revealed by the microscope. At times the motions seem quite normal. Thread-worms are not uncommonly present. The appetite may be poor, excessive, or fairly normal. Sometimes there is a history of recurrent abdominal pains of a colicky nature, and sometimes frequent attacks of acute indigestion.

(3) The last group of symptoms are those due to recurrent intestinal auto-intoxication. The child is not only pale and thin, but often looks wearied and has dark rings round his eyes. Many of the older children complain of headache and some of giddiness; and, although they may seem bright and intelligent, they easily get tired and irritable. Sudden attacks of pallor and faintness are not uncommon, and these are apt to be attributed by the patient's friends to heart-disease. In young children convulsions may even occur. Rarely, we meet with a degree of drowsiness, irregularity of the pulse, and vomiting, that are suggestive of tuberculous meningitis. Sleep is often restless and disturbed; night-terrors are common and, in older children, somnambulism. Grinding of the teeth and picking of the nose are often noticed, quite apart from the presence of intestinal worms, and enuresis is common. A very frequent and characteristic symptom is profuse perspiration; and in a few of the cases there may be a slight evening rise of temperature which may go on for weeks. The urine

is generally high coloured and strong smelling and shows a decided indican reaction. Sometimes a trace of albumin is present, and occasionally coli-bacilluria.

Diagnosis.—There is no class of cases more often misunderstood and wrongly treated by young practitioners—various mistakes are made. Sometimes the children are supposed to be in a general way “tuberculous,” and cod-liver oil is therefore given to them till they are sickened by it and put altogether off their food ; or the diagnosis is “debility,” and prolonged courses of iron tonics are administered with a somewhat similar result. Often, because the children are thin, an extra supply of “milk puddings” and other farinaceous foods is ordered to fatten them ; or, on account of their constipation, they are given large quantities of porridge and fruit.

Although this form of indigestion is often mistaken for tuberculosis, it must be remembered that many tuberculous children suffer from it also. When this is so, the treatment of the digestive disorder is important in addition to the open-air regimen. Fortunately, increased exposure to fresh air has a most beneficial effect on the digestion as well as on the tuberculosis.

Prognosis.—In considering the prospects of a speedy recovery we must take into account the degree of injudiciousness with which the child has hitherto been fed, and the extent to which the mother can, and will, carry out instructions. If the feeding has been on quite wrong lines and is at once altered according to directions, the result is in most cases strikingly successful ; the patient rapidly improves and “becomes quite a different child,” as the mother says. When the former dieting, however, has been fairly judicious, the improvement may be relatively slow. Should the reform in diet be carried out intermittently and in a half-hearted fashion, its effect is sure to be disappointing.

Treatment.—In starting the treatment of the case, the parents should be warned that it must be carried out strictly for some weeks at least, to begin with. When this preliminary period is past, there is usually no difficulty in persuading them to continue the diet, in spite of the trouble it causes, because the results are so satisfactory. The parents must also be made to realise that the matter is in their hands, and that the

medical man can only advise them as to the various details—dietetic, medicinal, and hygienic—by attention to which they are themselves to cure the child.

Dietetic Rules.—The exact details of the diet to be ordered in any given case must vary with the child's age and social circumstances, and also with the stage of the malady and its degree.

The chief points to remember are: *First*, that the child's ability to digest starchy food of all sorts, and sugar, is much less than that of normal children; and that, if too much of these foods is given, more or less severe auto-intoxication and discomfort will follow, often without any ordinary dyspeptic symptoms. *Secondly*, the usual incompletely digestible elements of the diet such as are found largely in fruits, vegetables, oat-meal, brown bread, milk-curd, and certain fatty foods, may greatly aggravate the child's condition and set up unpleasant nervous symptoms. In ordinary healthy children a great excess of any of these foods produces somewhat similar symptoms, but in these patients the morbid phenomena occur after very moderate amounts.

In framing a diet, the mother's experience of what the child has in the past been able to digest with comfort should always be taken into account. The directions given should *always* be written out; for, if this is not done, some of them are very likely to be forgotten and undesirable variations substituted for them.

The Table on p. 302, while it indicates the general lines to be taken in these cases, must, of course, be varied in individual instances.

The child should not be pressed to eat more than he is inclined for, and four small meals are better for him than three larger ones. Indeed, moderation, even in the food that seems to agree best, is always important, and he should never be allowed to take a very large amount of anything. He must also be encouraged to chew his food properly, and, with this in view, special attention should be given to the state of the teeth.

Even when the special diet is suiting him very well, it is important not to restrict the child's food too long, or too severely, because it is good for his digestion to be gradually trained to deal with those foods which give him some difficulty.

MAY NOT BE TAKEN.	MAY BE TAKEN.
<p>Ordinary bread, biscuits, porridge. Farinaceous puddings (sago, rice, tapioca, arrowroot, cornflour, etc.). Pastry, cheese, sweets of any kind.</p> <p>Much butter, any jam. Thick or vegetable soups. Fried or salt meats.</p> <p>Vegetables (except as opposite).</p> <p>Fruit. Plain milk.</p> <p>Cocoa, tea, and coffee.</p>	<p>Bread (baked hard in the oven), rusks. Malted foods ("Veda Bread," "Grape Nuts," "Force," etc.). Any of the malted "Infants' Foods," calf's-foot jelly, lemon sponge. A little butter or jelly. Clear soup, beef-tea, chicken-tea. Underdone meat, chicken, rabbit, fish, tripe, eggs (usually). A little cauliflower or spinach, or a <i>little</i> mashed potato. Fruit juice. Modified milk, whey, buttermilk, and milk with potash or lime-water. Milk prepared with peptogenic milk powder (with half the usual amount of cream, malted milk, or condensed or dried milk).</p>

Medicinal Treatment.—The medicines which are usually prescribed consist of alkalies, tonics, malt preparations, the so-called "intestinal antiseptics," and laxatives. Soda with rhubarb or nux vomica (Appendix E, Form. 19) is generally found useful; and, if there is much anæmia, iron may be cautiously tried—ferrum redactum or a laxative iron mixture (Appendix E, Form. 24); cod-liver oil is rarely well borne. Such "intestinal antiseptics" as salol are often given because of the offensive motions, but they are of little benefit; an occasional dose of calomel generally does good. A very useful drug, in our experience, is ichthyol, which may be given in doses of 1 to 3 gr. in capsules, or to younger children in solution (Appendix E, Form. 13). It is rare for children to object to its nauseous taste. Extract of malt does not usually agree well, though its laxative properties may be useful. Taka-diastase is frequently beneficial.

When constipation is present, liquid paraffin, cascara, compound liquorice powder, compound decoction of aloes, or Carlsbad salts may be tried. Abdominal massage and exercises to strengthen the abdominal muscles are helpful in many cases.

Hygienic Treatment.—Careful attention must be given to the child's clothing, as recurrent chills—for example, from

bare legs and arms—are apt to aggravate the symptoms. A cold douche, with suitable precautions, is usually good for these children; and widely open windows and *interesting* open-air exercise are always important. Over-exertion of any kind, however, is to be avoided, and emotional excitement, worry, and want of interest are all harmful. In the case of the younger and more nervous patients, a mid-day rest in a darkened room is a great advantage, even if the child does not sleep.

Complete change of air and scene do probably more good than any other single measure besides the diet. Many of these children, who are chronic invalids at home during school time, lose all their symptoms during the summer holiday, without any other special treatment. With some of them the seaside agrees very well, but hill air is preferable in most cases. The striking benefit derived from a country life is doubtless due in part to the increased amount of fresh air, but there can be little question that the change of scene and occupation and the mental stimulus of new experiences have also a great deal to do with it.

The Coeliac Disease

(*Acholia—Intestinal Infantilism*).

The name "Coeliac Disease" was given in 1888 by Dr Gee¹ to an interesting and important, though not very common, group of cases, the main features of which consist in obstinate recurrent diarrhoea lasting for years, a serious arrest of growth, pallor, muscular debility, and abdominal distension. The condition is now recognised as a clinical entity, and has been investigated and described under various names by a number of observers.² The accuracy of Gee's original description of the disease has been fully confirmed; but, so far, little has been added to our knowledge of its pathology, and its causation remains quite obscure, although we are undoubtedly better informed on the chemical changes which are present. G. F. Still, in his Lumleian Lectures,³ has given a good clinical

¹ S. Gee, *St Bart. Hosp. Rep.*, 1888, xxiv., 17.

² W. B. Cheadle, "On Acholia," *Lancet*, 30th May 1903, i., 1497; C. A. Herter, *Infantilism from Chronic Intestinal Infection*, New York, 1908; R. A. Gibbons, "The Coeliac Affection in Children," *Edin. Med. Journ.*, Oct. and Nov. 1889, 291 and 420.

³ G. F. Still, *Lancet*, 10th, 17th, and 24th Aug. 1918, ii., 163, 193, and 227.

description, to which and to Herter's monograph, as well as to the recent metabolic studies of Morris and MacRae¹ and of Leonard Parsons,² the reader is referred.

Clinical Features.—The coeliac disease is not due to poverty or to unhygienic conditions, for it occurs oftener in private than in hospital practice. It attacks the sexes practically equally, although if anything girls are more frequently affected than boys. The patients are usually, if not always, bottle-fed.

In most cases the earliest symptoms appear in later infancy—between nine months and two years. They may begin with an acute attack of diarrhoea, but in some cases there is constipation at first, in a few vomiting, and in others simple wasting. Sometimes the patients have formerly been healthy, sometimes they have suffered from digestive disturbances. Still found a history of infantile scurvy not infrequent.

Symptoms.—After the disease has gone on for a year or two the child's appearance is very characteristic. He is pale and listless, and, though his cheeks may still be round, his limbs are thin and flabby, and he is very easily tired and shows a strong and increasing disinclination to go upstairs. In one of Still's cases this flaccidity of the muscles was so extreme that the patient did not walk till he was more than six years old; and in most cases the children are unable to run or jump for a long time after the intestinal symptoms have been recovered from.

If the disease has lasted long, the small size and infantile proportions of the patient are very striking; he looks several years younger than he really is, and he seems always tired. On X-ray examination it is found that the stage of ossification in the limbs is that of the apparent, and not that of the real age, and that the bones are osteoporotic.

The mental development, though not defective in the usual sense, is often rather infantile, like that of the child's body; though his invalid life may have made him prematurely thoughtful and introspective beyond his years. He is, at times, touchy and irritable; and any excitement or mental effort is apt to be followed by obvious fatigue and depression. The voice is high-pitched and squeaky, probably from the larynx being abnormally small.

¹ N. Morris and O. MacRae, *Arch. Dis. Child.*, 1931, vi., 75.

² L. Parsons, *ibid.*, 1927, ii., 198.

The temperature-curve is normal, though it is sometimes a degree or so higher than usual.

The abdomen is more or less enlarged and feels doughy on palpation. This often leads to a mistaken diagnosis of abdominal tuberculosis. The liver is very rarely enlarged, and it almost always seems unusually small. Abdominal pain is rare, except in the later stages of the disease, and vomiting seldom occurs.

The condition of the stools is almost pathognomonic. To quote Gee's succinct phraseology: "Signs of the disease are yielded by the fæces; being loose, not formed, but not watery; more bulky than the food taken would seem to account for; pale in colour as if devoid of bile; yeasty, frothy, an appearance probably due to fermentation; stinking, stench often very great, the food having undergone putrefaction rather than concoction. The pale loose stool looks very much like porridge or gruel."

Chemical examination of the stools will usually reveal the excessive amount of fat which had been suggested by the naked-eye appearance. For the most part the fat is in the form of soaps and fatty acids. Fat-splitting is as a rule quite satisfactory and hence, because of the increase in the total fat, the proportion of neutral fat is usually diminished. Morris and MacRae, however, record a case in which for one period there was an abnormally high proportion (39·4 per cent.) of neutral fat.

Instead of the total fat forming 30 per cent. of the dried fæcal weight, as in health, the proportion in cœliac disease may rise to 60 per cent. and even 80 per cent. This increased proportion of fat in the fæces, however, is not peculiar to cœliac disease, as is often thought, since it may occur in simple diarrhœa and in abdominal tuberculosis. On the other hand, a true cœliac motion may contain only the normal proportion of fat, especially if the child is receiving a diet which contains little fat. But even with a normal fat intake Morris and MacRae record examples of active cœliac disease with motions containing only 26 per cent. and 32 per cent. of fat.

The most constant change in cœliac disease is a diminished retention or utilisation of fat. In no case during the active stage did Morris and MacRae obtain a normal retention of fat. In health over 90 per cent. of the fat ingested is retained. The

exact proportion, however, as shown by Hutchison,¹ varied with the intake, being greater the higher the intake. This holds good even with such a high intake as 290 grammes per day so long as there is no intestinal upset. In coeliac disease the percentage is much reduced, but here also as in health it is directly proportionate to the amount ingested. In the following chart from Morris and MacRae, in which the percentage absorption of fat is plotted against the intake, (a) the relationship between the amount ingested and the percentage absorption, and (b) the

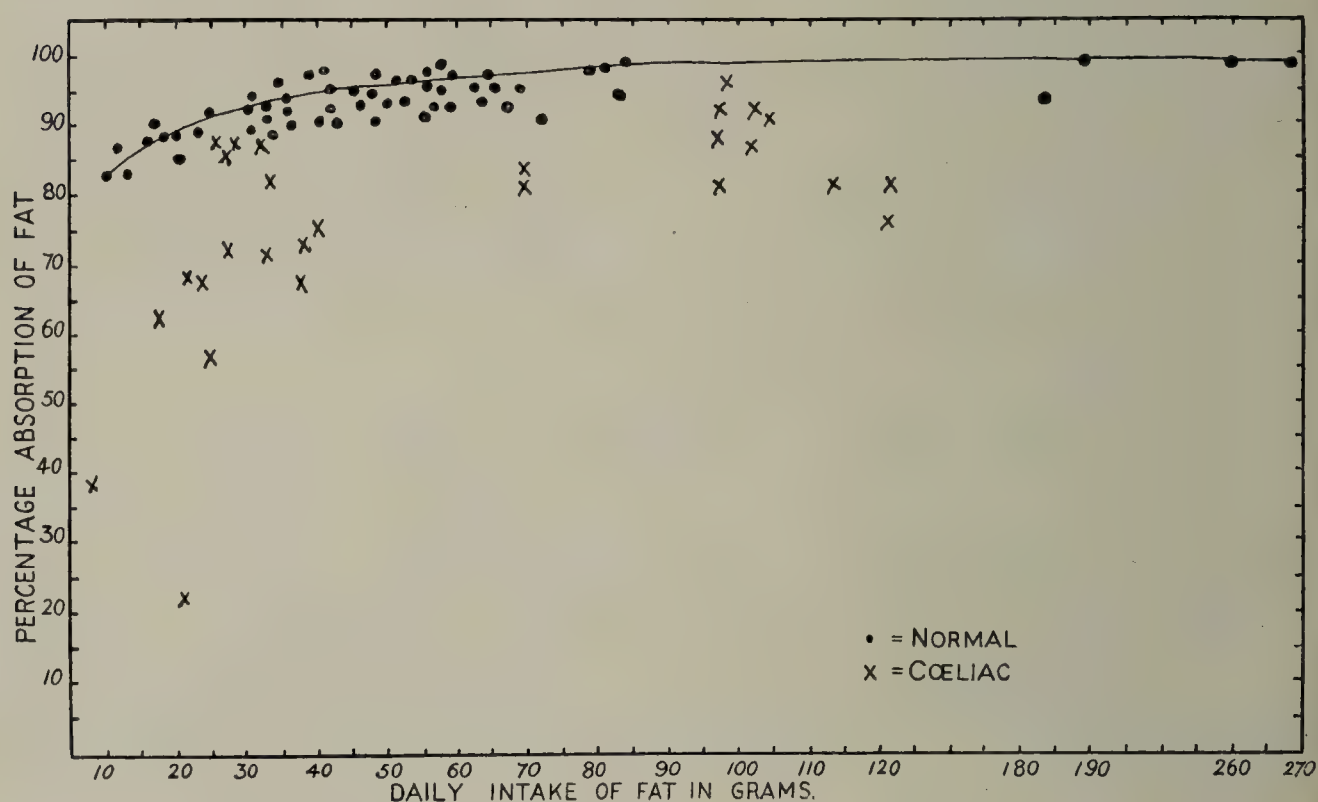


FIG. 97.—Showing percentage absorption of fat during different daily intake of fat in health and coeliac disease. (N. Morris and O. MacRae, *Arch. Dis. Child.*, 1931, vi., 75.)

reduced percentage absorption in coeliac disease, are well shown (Fig. 97).

Fat, however, is not the only proximate principle of the food whose absorption is impaired. As pointed out by Herter, protein also suffers, and Fanconi² and M'Lean and Sullivan³ demonstrated that the absorption of carbohydrate, as evidenced by blood-sugar curves, was interfered with. A low blood-sugar curve would seem to be pathognomonic of the active stage of coeliac disease.

In addition, the faeces contain an abnormal amount of the

¹ H. S. Hutchison, *Quart. Journ. Med.*, 1920, xiii., 277.

² G. Fanconi, *Beiheft. z. Jahrb. f. Kinderheil.*, 1928, xxi.

³ A. B. M'Lean and Sullivan, *Amer. Journ. Dis. Child.*, 1929, xxxviii., 16.

salts of calcium and phosphorus, due for the most part to their diminished absorption, but at times the excessive calcium in the fæces results from excretion by the bowel. At least this is the only conclusion which can be drawn from the fæcal output being greater in amount than that ingested with the food. This deprivation of calcium and phosphorus is reflected in the osteoporotic state of the bones, and in the development of rickets when the child recovers and growth reappears (p. 280).

Examination of the blood reveals a diminished serum calcium and serum phosphorus, the calcium as CaO varying between 6.3 and 7.8 mgrm. per cent. and the phosphorus as P_2O_5 between 2.0 and 4.7 mgrm. per cent., instead of the normal 10 and 11 grmm. per cent. and 4.0 to 6.0 mgrm. per cent. respectively. There may also be a degree of anæmia.

There is nothing characteristic in the state of the urine. Herter and Brown record an increase of indol derivatives.

A temporary improvement in the colour and consistence of the motions occurs from time to time, and occasionally they may for a short time look almost normal. Every few weeks there is an exacerbation, during which the motions become rather more frequent (three or four) and loose. The height and weight often remain stationary for months or years. Other symptoms, such as sweating on the head, coldness and clamminess of the extremities, and excessive or capricious appetite often occur.

In the later stages of the disease—after two years or so—the characteristic porridgy appearance of the motions is less noticeable and they become shreddy and soft, with a pale grey, greenish, or even a dark brown colour, and show an excess of mucus and sometimes a little blood or pus. These changes seem to indicate secondary inflammation in the bowel.

Complications.—As in other forms of long-continued diarrhœa, œdema and purpuric eruptions are not uncommon. Tetany occurred in three of Still's cases and scurvy in five. Rickets may develop during the stage of convalescence (Fig. 6, p. 281).

Prognosis.—The disease rarely lasts less than one or two years and often goes on much longer (three to eight). After the first two months the patient settles down to an invalid existence, periods of slight improvement being succeeded by frequent relapses. Later, in those cases which do well, the stages of improvement become more definite and last longer,

and the relapses are fewer and less serious; weight is gained, an ounce or two at a time; and, finally, the appetite improves and the motions become more constantly normal, in spite of the diet being more ordinary. Relapses are common and sometimes serious, and they may end fatally. Complete recovery of health takes place in a considerable majority of those cases which are carefully and continuously treated, but probably only in a small minority of those in which the diet is not properly regulated and too much reliance is placed on drugs. In our experience, however, as in Still's, the patients in the long protracted cases have always remained subnormal in stature. Death may occur from a severe relapse of the diarrhœa, or it may be due to intercurrent disease.

Morbid Anatomy.—Very little that is distinctive has been found post-mortem. There may be atrophy of the bowel and of the viscera in general, but the latter at least is most probably the result of the concomitant starvation rather than the cause of the condition. The catarrhal and ulcerative conditions of the bowel sometimes observed are also considered secondary phenomena.

Causation.—Although some abnormality of the bowel would appear the most probable cause of this affection, all observers, including Gee, have failed to discover any sufficiently marked or characteristic change in the intestine.

Cheadle, who also appreciated the absence of any definite pathological change in the gut, concluded, but erroneously, from the colour of the stool, that there was an absence of bile and hence suggested the term *acholia*. And since jaundice was not present he postulated an arrest of the cholegenetic function of the liver, probably in response to reflex action from difficult teething. We now know that there is not an absence of bile or bile salts in the cœliac stool, although the pallor might suggest such. But, as Gee says, "the colour of fæces is a very rough measure of the quantity of bile which they contain." In opposition to the idea that acholia plays any part is the fact that in such a condition as atresia of the bile ducts the metabolic picture is quite different. In these circumstances phosphorus is absorbed quite well, as the bulk of it appears in the urine, whereas in cœliac disease it is *via* the fæces that the loss of phosphorus occurs. And further, in biliary atresia the blood-sugar curve is quite normal. Nevertheless, it is held

by some writers to-day that there is a deficiency of bile salts and they record improvement by their administration. The evidence on this question is, however, conflicting.

Gee in his original description remarks that "sometimes from India Englishmen return sick with the cœliac affection." He is, of course, as Still says, referring to sprue, and certainly there are many resemblances between the sprue of adults and the cœliac disease of children. It was probably the reputed association of tropical sprue with dysentery which suggested to Still and Nabarro their bacteriological investigations, which enabled them to isolate the dysentery bacillus from a large proportion of their cases. This view regarding the etiology, however, has not been generally accepted. Herter suggested that the cause was "an overgrowth and persistence of bacterial flora belonging normally to the nursing period." The prevalent organisms found in the gut at this age are the Gram-positive *B. bifidus* type, the *B. infantilis* type, and the coccal type. Alan Brown¹ and his co-workers have confirmed Herter's findings of the infantile type of flora in "chronic intestinal indigestion." These workers detected the presence of another organism—a spore-bearing bacillus (Bacillus A), which has a proteolytic effect and increases the ammonia content of the fæces. This change in reaction is considered by Brown an important factor in the pathogenesis of cœliac disease.

Morris and MacRae conclude that the disease is defective intestinal absorption and "due to a change or changes in the physico-chemical constitution of the intestinal contents." The beneficial effects which Brown obtained with the administration of lactic acid cultures, and those obtained by Morris and MacRae, not only in a better absorption of fat but also of calcium and phosphorus, during the ingestion of the acid phosphate of soda, lend definite support to such a theory.

The defective absorption from the gut has been attributed to an over-rapid passage of the intestinal contents, and Freise and Jahr² claim that they were able to obtain better absorption by slowing the passage of the chyme through the administration of opium and atropin. Morris and MacRae, however, were not able, either by the administration of carmine or

¹ A. Brown, A. M. Courtney, *et alia*, *Amer. Journ. Dis. Child.*, 1925, xxx., 603.

² R. Freise and J. M. Jahr, *Jahrb. f. Kinderheil.*, 1925, cx., 205.

charcoal, or by means of X-ray examinations after a barium meal, to detect any increased rapidity in the passage of the food through the bowel.

M'Carrison¹ has suggested that coeliac disease may possibly be due, to some extent, to deficiency of vitamins in the diet, as he has found similar symptoms occurring in pigeons fed exclusively on autoclaved, milled rice.

Treatment—Dietetic.—Success in the treatment of coeliac disease depends very little on medicines; and we have to rely almost entirely on extreme care and discretion in managing the details of the diet and hygiene, and suiting them to the idiosyncrasies of the individual child. There are very few diseases, indeed, which try the patience and perseverance of all concerned so severely. Our great aim is, of course, to improve the patient's nutrition, and especially to increase the defective fat-assimilation as far as is possible. This we endeavour to do by giving the necessary amount of fat only in such forms as the child has been found able to digest. Another indication which must be borne in mind is the lessening of unabsorbed decomposing fat in the bowel. To this end we must reduce the amount of fat in the food as far as we can without dangerously reducing its nutrient value. As this invariably means under-feeding, the child should be confined to bed in order to conserve his energy and to guard against chill.

In considering in what respects the diet is to be restricted, we find that most of the common foods that agree with healthy children have to be forbidden. Fresh cow's milk must be greatly lessened, or if possible stopped altogether, for it is always found to disagree badly in these cases, but skimmed milk is well borne. Apparently the fat in human milk is well tolerated, as Still in two serious cases obtained better results with it than with anything else. The good results obtained may be due to the different reaction of the intestinal contents on a diet of human milk. Dried milks frequently agree quite well even when they contain the normal amount of fat.

In a few cases in which cod-liver oil can be taken it is very beneficial, and Holt² has published a case in which it

¹ R. M'Carrison, "Pathogenesis of Deficiency Disease," *Indian Journ. Med. Research*, 1919, vi., 3, 275-355, and 1826-1827, vi., 4; also Abstract in *Brit. Med. Journ.*, 12th July 1919, ii., 39.

² Holt, Courtney, and Fales, *Amer. Journ. Dis. Child.*, 1917, xiv., 2, 222.

had a remarkable effect on the retarded growth. This was almost certainly not due to a particular variety of fat but to the accompanying vitamin D, which has a definite effect on mineral absorption. Radiostol would give equally good results.

The diet should consist at first of a maximum of protein and a minimum of fat and carbohydrate. Even moderate amounts of sugar or other carbohydrate almost invariably cause fermentation with an exacerbation of the symptoms. In the severe cases, a period with limitation to protein milk (two or three pints per day according to the age of the child) should be commenced with. Later, skim milk (fresh or dried), junket, jellies, and sponges should be given. To this may be added at a still later period shredded meat, chicken, and fish, but the meat must be lean and the fish white (cod, haddock, or whiting) and steamed. The meat can be given as a raw meat sandwich between very thin slices of stale bread. When the motions have become less bulky, more or less formed, and darker in colour, carbohydrates in the shape of rusks, tea biscuits, or rice boiled in water may be carefully tried. Water *ad libitum* can be given to drink, but a moderate amount of skimmed milk (one to one and a half pints per day) should be continued throughout the treatment.

In dieting these children one should proceed from the spare to the more liberal diet very slowly, only trying one change of article and that in small quantity at a time.

Medicinal Treatment.—The drugs which have the greatest effect in increasing absorption of fat and minerals are the acid phosphate of soda and bile salts. Taurocholate of sodium and glycocholate of sodium can be given in $\frac{1}{4}$ or $\frac{1}{2}$ grain doses of each three times daily. Allen and Hanbury prepare an elixir containing $\frac{1}{2}$ grain of each salt in half a drachm. Radiostol, while increasing the absorption of the calcium, rather impairs absorption of fat. Pancreatic extract and thyroid extract, which have both been tried, are useless.

If a purgative is indicated, castor oil is the best. Apart from its purgative action, the regular administration of 5 minims thrice daily may be followed by improvement in the stools. This may be due to it exerting a constipating effect in this dose, and thus slowing the passage of the intestinal contents.

Small doses of brandy are beneficial in some cases.

Hygienic Treatment.—While carefully and continuously regulating the diet, we have to attend to the hygienic conditions and do all we can to shield the patient from exhaustion, and from anything that might favour complications. Warm clothing is necessary, with open air short of chilling, if possible in a sunny equable climate. While the symptoms are progressing, the child should not be bothered with lessons, but congenial occupations and regular exercise, which must always stop short of fatigue, are indicated. Herter emphasises the value of soothing human surroundings and companionship. From what has been said, it is easily seen that the means at our disposal at present for the treatment of this disease are far from being satisfactory; but yet, as Still says, “it is nearly always possible to obtain marked improvement in these cases, and a large proportion of them can be coaxed back, after many months or years of patient perseverance, with a very restricted diet, to health and even to an average power of assimilation.”

Duodenal and Gastric Ulcers.

Duodenal and gastric ulcers, comparable to those commonly seen in adults, are very rare in childhood, and are met with practically only in older children. Many instances of duodenal ulcers in young babies have, however, been placed on record¹; and, as the symptoms are generally obscure, it is probable that the condition often escapes recognition. The ulcers may be single or multiple. They are usually situated on the back wall of the bowel above the papilla.

In fully a third of the cases, according to Holt, there are no definite symptoms. In some, there are indications of perforation and peritonitis—although these are often not easy to make sure of; in a few, evidence of pyloric spasm has been noted. In the large majority of cases, however, the only definite symptom is hæmatemesis or melæna.

Seeing that ulcers have been found cicatrised in several cases of infants who have died of other diseases, it is evident that their spontaneous recovery may take place, though it rarely does so. The medical treatment must be carried out on

¹ Henoch, *Lectures on Children's Diseases*, New Syd. Soc., i., 67; L. E. Holt, *Amer. Journ. Dis. Child.*, 1913, vi., 381; Donald Paterson, *Lancet*, 1922, i., 63.

general lines ; and surgical procedures are rarely justifiable. One successful operation has, however, been recorded.¹

Lienteric Diarrhœa.

Lienteric diarrhœa is an ailment which should always be borne in mind when recurrent colic and loose motions occur in an otherwise healthy child. When the case is recognised to be of this nature it can almost always be quickly cured by appropriate treatment ; while, if it is mistaken for one of the ordinary forms of dyspeptic diarrhœa, as often happens, it may last a long time and give a great deal of trouble.

Clinical Features.—The cardinal symptoms of the malady are three in number—sudden discomfort, griping pains, and an urgent desire to empty the bowel. These set in during, or immediately after, the taking of food, and often the child has to rise hurriedly from the table when he has scarcely begun his meal. The motions consist of half-digested food with much mucus. In a few cases the discomfort and pains occur without diarrhœa following. When the motions are large the child may become weak and nervous from loss of nourishment ; but in other respects he usually seems fairly well.

Lienteric diarrhœa is most commonly met with in children between five and eight, but it may occur at any age. It is important to remember, also, that the condition is occasionally met with in quite young babies. In them, owing to the frequency of the feeding, the relation of the diarrhœa to the taking of food is apt to be overlooked.

Treatment.—The symptoms are not usually influenced at all by restriction of the diet, and astringent diarrhœa mixtures have no effect on them. They are brought to an end at once, however, by small doses of liquor arsenicalis. In older children the dose is one drop, which should be given with a few drops of tincture of cardamoms in a teaspoonful of water immediately before each meal. In babies, half or even a quarter of a drop, before each bottle, is usually enough. If the case is obstinate, 2 or 3 grs. of Dover's powder may be given to older children ; but generally the arsenic is entirely successful.

¹ D. W. Palmer, *Annals of Surgery*, 1921, lxxiii., 545.

Intestinal Worms.

The chief intestinal parasites met with in children in this country are thread - worms, round - worms, and tapeworms. Thread-worms are much the commonest variety; tapeworms are rare in children under four years.

The presence of worms can only be recognised with certainty by finding either the parasites or their ova in the stools. In doubtful cases eosinophilia should be looked for. Their treatment always requires close attention to details and sometimes great perseverance.

Thread-worms (*Oxyuris vermicularis*).—Thread-worms live mostly in the cæcum and appendix; but they are also found in other parts of the colon, in the rectum, and, less frequently, in the small intestine. The ova are taken in by the child along with his food; and they are apt to be constantly reintroduced by his fingers, which become contaminated owing to his scratching the neighbourhood of the anus.

The general symptoms are mainly those of chronic intestinal catarrh, but sometimes nervous symptoms such as convulsions are also met with. Local symptoms of various kinds, due to the irritation of the worms, may also be present. Among these are severe itching about the anus, mucous diarrhœa, tenesmus, prolapse, frequent micturition, retention of urine, priapism, and vulvar discharge. The nostrils, also, are sometimes red, itchy, and sore from the child's picking his nose with his fingers soiled with the irritating substance of the thread-worms.

Treatment.—To be successful, the treatment must aim not only at expelling the parasites, but also at preventing reinfection and improving the state of the bowel.

Expulsion of the Worms.—To drive the worms down into the lower part of the bowel, a dose of castor oil or of calomel and santonin (āā 1 gr.) is first given. When this has acted, the colon should be cleared by a large soap-and-water injection. It is well to use a vermicide enema after this, and to repeat it every alternate morning for a week. For this purpose, a solution of common salt (a teaspoonful to 5 oz.) or infusion of quassia (5 to 7 oz.) may be given after the bowels have acted. The fluid is to be introduced slowly and allowed to stay in as long as possible. In inveterate cases we have seen good results follow daily injections of emetine (gr. $\frac{1}{4}$) extending over a week.

Prevention of Reinfection.—This is most important, and it is often forgotten. The anal region must be carefully washed after each motion, and it should be smeared with ung. hydrarg. The child's nails must be kept very short, and frequently brushed with carbolic soap. Any uncooked vegetable food that he takes should be carefully washed.

Improvement of the General Health and of the Digestion.—Both general debility and indigestion strongly favour the continuance of the worms. The treatment is therefore not complete until the general health and digestion have been attended to. Iron or bitter tonics may be required, and the diet may have to be regulated on the lines recommended for chronic intestinal dyspepsia (p. 301).

Round-worms (*Ascaris lumbricoides*).—These live mostly in the small intestine, but wander at times into other parts of the alimentary canal. The ova are swallowed in impure water.

The symptoms are usually indistinguishable from those of dyspepsia. Sometimes nocturnal diarrhœa occurs. Convulsions, sudden rises of temperature, and other nervous symptoms are not uncommon. Frequently, however, there are no evidences of illness, the appearance of the worm in the motion being the only disturbing factor.

Treatment.—This consists in the administration of santonin, alone or combined with calomel (āā 1 gr.). The powders may be given every night for three nights, and should be followed in the morning by a dose of senna or salts.

Tapeworms.—The *Tænia mediocanellata* or beef tapeworm is much the commonest species in Britain; though the *T. solium*, acquired from pork, is sometimes found. While these species are too well known to require description here, attention may be specially called to the *T. cucumerina*, which is occasionally found in young children who are in the habit of playing with dogs. This tapeworm is small and short (6 to 12 inches), and its proglottides are long and narrow and have bilateral genital pores; they are mostly separate when passed, and look like rather large flat grains of boiled rice. The larval form develops in the louse of the dog, and is carried to the child's hands or to his food by the animal's tongue.

Treatment.—There is sometimes very great difficulty in getting rid of tapeworms in children; and even when the

details of treatment are carefully attended to, the head is often left in the bowel. The following measures have been found successful in obstinate cases: (1) A number of small capsules containing extract of male-fern should be prepared, and also similar capsules containing treacle. For a week or two the child should be trained daily to swallow the treacle capsules till he can do so quite quickly and easily.

(2) He should then be put to bed for two days and given only such articles of diet as will lessen the intestinal contents and the secretion of mucus in the bowel—soups, jellies, eggs, Benger's and Mellin's Foods. He should have no milk and no farinaceous food.

(3) During these days his bowels should be kept moving by a daily dose of citrate of magnesia or other mild laxative.

(4) On the second night, a dose of castor oil should be given and no food after it until the worm has been passed.

(5) On the third morning he should have, at intervals of an hour, three doses of the male-fern capsules, each equivalent to 30 minims.

(6) Three hours after the third dose of capsules, castor oil should again be given. Capsules sufficient for four doses must be prepared in case some of those given should be vomited.

The **Whip-worm** (*Trichocephalus dispar*) is often found post-mortem in the intestine in children, and, during life, the ova are not uncommon in the stools. It produces no symptoms and no drug seems to have any decided effect upon it.

Acute Intussusception.

This is a most important, as well as characteristic, disease of infancy, as it accounts for fully three-fourths of the cases of acute obstruction of the bowels in young children. It occurs frequently in breast-fed babies and is commonest between four and eight months; a considerable majority of the patients are under one year. Older cases, however, are not uncommonly met with.

Symptoms.—The onset of the disease is almost always quite sudden; although the acute symptoms may have been preceded by a few days of gastro-intestinal derangement. Occasionally there is a history of a fall or other forcible movement of the body having occurred immediately before.

The main symptoms are three in number: (a) abdominal

pain; (*b*) vomiting; and (*c*) the passage of blood and mucus with straining.

The *pain* is nearly always the first thing noticed. It has the characters of severe colic and is accompanied by loud screaming and kicking. It goes on recurring at short intervals for a varying period, and then, after perhaps twenty-four or thirty-six hours, paralysis of the bowel sets in and gangrene threatens. The patient gradually becomes collapsed and drowsy, and the spasms cease to return.

The *vomiting* is severe and repeated, and generally occurs immediately after the pain begins, though occasionally it is noticed first.

The *passage of blood and mucus from the bowel with straining* is rarely absent when the patient is a baby. It sometimes follows immediately after the other symptoms, but usually there is an interval of some hours. The duration of this interval depends on the situation of the intussusception (being later when the small intestine is involved) and on the degree of congestion of the affected parts. Generally at the first onset of the symptoms there is a forcible discharge from the bowel of any fæces it may contain; and thereafter nothing comes but blood and mucus.

Physical Signs.—The patient is often a fat, well-nourished baby, and in the intervals between the pains he looks fairly well, though there is always some collapse and the pulse is rapid. The degree of collapse varies greatly in different cases; sometimes it is extreme, and the patient presents a typically abdominal facies. At first the child may appear absolutely well between the attacks, and unless a thorough examination is made the condition will be missed. The temperature is normal or subnormal at first. If there is any fever, it usually indicates the presence of inflammatory complications.

The abdomen is generally soft and not much distended, and moves freely with respiration. There may, however, be considerable tenderness and rigidity of the abdominal wall, in which case an anæsthetic may have to be given before a satisfactory examination can be made.

In the great majority of cases, though not always, a firm sausage-shaped tumour is felt on careful palpation. It may be situated almost anywhere in the abdomen, the exact position depending on how far the invaginated portion of bowel has

travelled; often it feels quite superficial. It varies considerably in different cases in size and in mobility as well as in position and hardness. Sometimes during palpation the tumour may be felt to alter in consistence owing to recurrent muscular contractions. Usually the right iliac fossa is felt to be noticeably empty. In intussusception of the small intestine the tumour is generally more difficult to make out. It is now believed that intussusceptions begin far more frequently in the lower part of the ileum than used to be thought, and less frequently at the ileo-cæcal valve. This part of the bowel, owing to its thicker mesentery, is specially liable to gangrene.

The lower end of the intussusception may often be felt per rectum, and the examining finger when withdrawn is found to be covered with blood. Even when the intussusception cannot be felt in this way, it may be possible to palpate it easily on bimanual examination. Occasionally the invaginated bowel is protruded through the anus, but the diagnosis should always be made before this occurs.

In older children the symptoms of intussusception are often less definite, and the diagnosis, therefore, is more difficult. The onset may be less distinct and the tumour difficult, or even impossible, to find; and there may be no passage of blood or mucus from the bowel. The obstruction, also, may not be complete, and fæcal matter may therefore continue to be passed.

Diagnosis.—If a clear account of the onset of the attack is obtained, the diagnosis is usually easy, even when the tumour cannot be very easily made out. In most cases, however, the sausage-shaped tumour can be recognised without much difficulty under an anæsthetic, and often without.

Sometimes a severe acute ileo-colitis is taken for an intussusception, but this mistake should not often be made. The onset in the former is not so sudden, the vomiting is less urgent, bile-pigment is much more likely to be found in the stools, the blood in them is much less in amount and also more thoroughly mixed with the fæces, there is no tumour, and the temperature is usually raised. It is, however, possible that the two conditions may coexist.

Occasionally, though very rarely, cases of *incomplete or chronic intussusception* occur.¹ In these the clinical history is apt to be

¹ "On Chronic Intussusception in Children," G. F. Still, *Arch. Ped.*, March 1921, xxxviii., 174.

misleading, because there has been an almost complete absence of the most characteristic of the usual symptoms. Although the illness has begun as usual with abdominal pain and vomiting, these have often not been very severe, or have not been thought to be so, and they have recurred at longer intervals. The stools have either shown no blood at all or what there has been was not more than many children have from a slight local lesion; there has often also, at first, been no obstruction at all—only a degree of constipation. After a time the vomiting and colic tend to lessen and the intervals between them to become longer. The child, however, always goes on getting thinner and weaker, and obstruction ultimately sets in.

As the symptoms are so much less urgent than in the acute cases, the patient is often not seen for several days or even weeks after they have begun and the parents are becoming anxious about the child's steady wasting. In such long-standing cases the tumour is sometimes very hard and may resemble a mass of tuberculous glands. In rare cases, in older children, the invaginated portion of gut sloughs and is passed with the motions, and the child slowly recovers.

Course and Progress.—The local inflammation set up in these chronic cases is usually much less severe than in the acute ones, and the intussusception is therefore reducible by operation even several weeks after the onset of the symptoms. In ordinary acute cases of intussusception, spontaneous reduction occasionally occurs in the early stages. Spontaneous recovery, however, is so uncommon that it can never be expected. If it does not occur, and the invagination is not successfully relieved, the child soon passes into a state of collapse and dies, in most cases, within four or five days, if not sooner.

Treatment.—There are two methods of treatment which have proved successful in intussusception: (1) distension of the bowel by water or air introduced by the rectum; and (2) laparotomy, with reduction of the intussuscepted bowel by direct manipulation.

1. *Distension of the bowel* from the rectum may be effected by warm water from an ordinary douche apparatus or a Higginson's syringe, but inflation with air by means of a small bellows is more effectual. The child must be fully anæsthetised, and while the air or water is being introduced,

the nates should be firmly compressed to prevent return, and the abdomen carefully palpated. The proceeding must be carried out slowly and deliberately, and the distension continued for some time. There is considerable danger that the intussusception may be only partially reduced, and may recur. If complete reduction has been effected, a fæcal motion usually occurs shortly after, and the child seems greatly relieved.

2. *Laparotomy*.—The details of the operation need not be considered here. It may, however, be mentioned that the reduction is always to be attempted by manipulating the apex of the intussusception through the sheath, and pressing it up from below, and never by pulling on the bowel above it. When reduction is found impossible, the tumour must be excised and the cut ends of bowel united, or an artificial anus may be made. In these latter circumstances, however, the chance of recovery is extremely small.

Choice of Treatment.—Many years ago when the mortality from laparotomy in such cases was very great, even in the hands of distinguished hospital surgeons, the unsatisfactory proceeding of distending the bowel from the rectum was usually preferable, because it offered a better chance of recovery. Now, the progress of surgery has changed this state of things entirely, and there cannot be any doubt whatever that immediate operation by a competent surgeon should always be done without delay. In proof of this it need only be mentioned that out of twenty-four consecutive cases of acute intussusception operated on by Sir Harold Stiles in the Royal Edinburgh Hospital for Sick Children within thirty-six hours of the onset of symptoms, twenty-three recovered (*i.e.*, 95 per cent.).¹ The early stage at which the disease is now generally recognised and handed over to the surgeon is a very important factor in the good surgical results obtained to-day.

The drawbacks to the treatment by distension of the bowel are many and important. At best, it can only be expected to succeed in very recent cases; for though an intussusception has been reduced in this way as late as the seventh day (Cheadle), this is quite an exceptional experience, and even by the end of twenty-four or thirty-six hours the intussuscepted portion of gut

¹ Full details of the cases of acute intussusception operated on in the Edinburgh Children's Hospital up to May 1906 will be found in a paper by Henry J. Dunbar, *Scot. Med. and Surg. Journ.*, August 1906, xix., 97.

will probably have become so much swollen from œdema as to be irreducible. Should the lesion be situated above the ileo-cæcal valve no effect can, of course, be expected from inflation.

Although reduction by the injection of air or water should not be thought of when competent surgical assistance is available, it should not be forgotten under other circumstances. It has the advantage that the necessary apparatus is always obtainable, and that its use requires only ordinary sense and tact. It may also be said that if these are used, and the reduction is not effected, no harm has been done, and when the surgeon does arrive the operation may still succeed. Distension, moreover, when practised early is quite often successful. Our own experience of it is small, being confined to three cases which were under our care many years ago. In one of these—a partial case in an older child—the intussusception was not completely reduced, but recovery took place ultimately, portions of the invaginated bowel having sloughed and been passed per anum. The other two cases, which occurred in strong, healthy babies, and were dealt with at an early stage, were completely successful. In all three air was used, though in one, or two of them, water was first tried unsuccessfully.

Appendicitis.

Appendicitis may occur even in breast-fed babies of a few months, and it is not rare at any period of childhood. The symptoms in children differ in no important respect from those in adults, and perforation and gangrene often take place.

Symptoms.—The onset is generally acute, severe abdominal pain with tenderness on pressure on the abdominal wall over McBurney's point, vomiting often with diarrhœa, a foul tongue, and fever are the first symptoms in most cases. The local hyperæsthesia of the superficial tissues is an extremely important symptom. It is generally found in the right iliac or lumbar region, but may also be equally distinct elsewhere, and the possibility of a pelvic appendix must always be borne in mind. The pulse is usually rapid (120 to 140). Sometimes obstinate constipation is present, and occasionally tenesmus. Bladder symptoms and rectal tenderness are common; they are also met with, however, in cases of pelvic abscess, and in general pelvic peritonitis. The temperature keeps up and the vomiting usually continues, and within a few days, in an advancing case,

some inflammatory induration can often be made out in the right iliac or lumbar region. In some severe cases with gangrene the symptoms are very slight.

Diagnosis.—In all cases of acute abdominal disease with local tenderness, quick pulse, and vomiting, appendicitis should be suspected. If there is constant pain, and most of all if there is distinct tenderness on pressure over McBurney's point (or even elsewhere in the lower half of the abdomen), this is strongly in favour of the diagnosis. The presence of bladder symptoms is also confirmatory. A rectal examination should always be made in any doubtful case and is often of great value, as in this way alone a pelvic appendicitis may be recognised.

Among the diseases most often mistaken for appendicitis are the following: acute general peritonitis from pneumococcal or streptococcal infection; acute exacerbations of tuberculous peritonitis, such as not infrequently occur from the rupture of a mesenteric gland abscess; acute entero-colitis (which indeed is not very rarely present along with the disease of the appendix); intussusception, and typhoid fever. Strangulation by a Meckel's diverticulum is a rare condition, but when it does occur its symptoms may be indistinguishable from those of appendicitis. Occasionally simple impaction of hard fæces may also simulate this disease.

Acute pleuro-pneumonia of the right base sometimes gives rise to abdominal pain and acute tenderness very like those of appendicitis. In young children severe attacks of gastric influenza, of Henoch's purpura, and of cyclic vomiting, may also, in their early stages, be difficult to distinguish from it. In girls, about the time of puberty, symptoms suggestive of subacute appendicitis are sometimes caused by ovarian disturbances. The tenderness in these cases is generally situated lower and is present on both sides. It must be remembered, however, that tenderness of both ovaries may be present in genuine cases of appendicitis.

Treatment.—In watching a doubtful case, it is most important *to abstain from giving opiates*, which mask the symptoms; also, especially *to avoid purgatives*, which greatly increase the risk of perforation; and, lastly, to give no food for some time. Acute perforating cases are so common in children that, when the diagnosis is made, the necessary operation should always be done with the least possible delay.

Occasionally a mild form of catarrhal appendicitis occurs with influenza. When this occurs it is advisable that no operation should be performed during the acute stage, as the presence of influenza predisposes strongly to the occurrence of secondary infections.

Pneumococcal Peritonitis.¹

Pneumococcal infection of the peritoneum is commoner in children, especially in young children, than it is in adults. In them also it is more frequently found associated with other pneumococcal lesions, such as those of the lungs, pleuræ, pericardium, meninges, joints, subcutaneous tissue, and ears. Apart from those due to appendicitis and to tuberculous lesions, the large majority of the cases of acute peritonitis met with in children are of this nature.

Cases of pneumococcal peritonitis are either (A) *Primary*, that is to say, those in which the peritoneal inflammation is the original lesion; or (B) *Secondary*, in which a previous pneumococcal infection in the lungs, pleuræ, or elsewhere, has spread to the abdomen. The primary cases may be either acute or chronic, according to the intensity and course of the disease. They, unlike the secondary cases, are mainly seen among the lower classes. The secondary cases are more often subacute than the others.

Clinical Manifestations.—(A) In the worst of the *acute primary* cases, the onset is very rapid, with severe abdominal pain, persistent vomiting, and great prostration, which are soon followed by extreme drowsiness and delirium. The tongue is coated and the breath foul, and sometimes there is an eruption of labial herpes. The facies is that of abdominal disease (Fig. 8, p. 10). The temperature is high and irregularly intermittent from the first, the pulse feeble and rapid and there is a high degree of leucocytosis; the abdomen is rigid and retracted in its lower half. When the peritoneum is opened, a varying amount of brownish green, flocculent, sticky fluid of a watery consistence, and devoid of fæcal odour, is found in the lower part of the abdomen. It is usually about the fourth day before the exudate becomes distinctly purulent. Generally these cases are rapidly fatal. In many of the acute

¹ H. Rischbieth, *Quart. Journ. Med.*, Jan. 1911, iv., No. xiv., 205; J. E. McCartney and John Fraser, *Brit. Journ. Surg.*, 1922, ix., No. 36.

cases, however, the symptoms, although severe, are not quite so violently acute.

In an ordinarily severe acute case we may distinguish, to begin with, an *introductory stage*, in which the infection is localised in the pelvic peritoneum; and, within a few days, this is succeeded by a *second stage*, which is one of general blood infection—septicæmia. This second stage is easily recognised by the drowsiness and other general symptoms. If it lasts any time, the children rarely recover. This division of the symptoms into two distinct stages is of importance in the treatment, as well as in the diagnosis and prognosis.

In the *chronic primary cases*, which are much less common than the acute ones, the illness begins with colic-like pains in the lower segment of the abdomen, followed by profuse diarrhœa and recurrent attacks of vomiting. These symptoms may continue for a week or two, without the child seeming very ill, but she becomes nervous and exhausted and loses flesh. Gradually, after a time, the vomiting stops, but the colic continues and there is frequent and painful micturition. The temperature keeps up, the pulse remains rapid, and the abdomen becomes swollen and tender. When the abdomen is opened, an encysted pneumococcal abscess is usually found.

(B) In the *secondary cases*, the child usually has a history of having recently recovered from an attack of pneumonia or some other pneumococcal lesion and of having begun, after being apparently well for a time, to complain of abdominal pains, with a return of the fever, vomiting, and prostration. Laparotomy, in these cases, shows a subacute pneumococcal inflammation of the peritoneum, usually in the upper part of the abdomen. These cases not infrequently recover.

Etiology.—In cases secondary to other pneumococcal lesions, this is usually easy to understand. In the primary cases, which are all in the female sex, McCartney and Fraser have shown that the disease begins as a pelvic peritonitis; and that the infection probably always comes from pathogenic pneumococci in the vulva which spread from thence into the peritoneum through the vagina, uterus, and Fallopian tubes. The differences in the clinical characters in different cases probably depend on variations in these organisms.

In the great majority of cases the girls affected are between 6 and 7 years old. This is probably to be attributed to the

facts that, before that age, the lumen of the internal passages is closed; and that, after it, the reaction of the vaginal secretions changes from alkaline to acid.

Diagnosis.—Cases of pneumococcal peritonitis differ from those of appendicitis in the absence of the local signs characteristic of the latter disease.

In secondary cases, the nature of the attack can usually be suspected from the history of a recent attack of pneumonia or empyema.

In the acute cases, the very early onset of the vomiting, its extremely severe character, and the history of high fever from the beginning, are in favour of this form of disease; the severity of the diarrhoea is also characteristic. The rapid change in the symptoms which occurs when septicæmia begins, and its early onset, are also more striking in these cases than in most others.

In the chronic cases, the symptoms are occasionally mild and show little pain or tenderness. In such cases, a diagnosis of abdominal tuberculosis is apt to be made. In young children, a negative tuberculin reaction may be helpful.

Prognosis.—Pneumococcal peritonitis is usually a serious disease, and in its most acute forms generally fatal. A considerable number of the less violent acute cases, however, recover satisfactorily under surgical treatment. Subacute cases with abscess formation often get well when the pus has been evacuated and the cavity drained. A few mild subacute or chronic cases have been known to recover spontaneously.

Treatment.—The usual surgical procedure in these cases consists in the free opening and draining of the peritoneal cavity. McCartney and Fraser recommend that, in acute cases, this should always be done at once, under gas and oxygen; and they have found that their results have improved greatly since they have made a regular practice of also transfusing the patient with the parent's blood. To be useful, the transfusion must be done at the time when the evidences of septicæmia are just beginning to appear—earlier or later than this, it is of little value. In a child of 6 years, they aim at giving 250 c.c. of blood. The effect of the transfusion is often immediate and most striking; “the cyanotic tinge disappears and is replaced by a healthy rosy colour, the restlessness abates, the pulse rate slows, and the patient often drops into a sound sleep.”

CHAPTER XIV

CONGENITAL MALFORMATIONS AND DERANGEMENTS OF THE ALIMENTARY TRACT

Congenital Malformations of the Œsophagus

Congenital Atresia.—When an apparently normal baby is found, soon after birth, to be unable to swallow, and that even a teaspoonful of food immediately sets up coughing with cyanosis, and, mixed with much mucus, is returned at once, a malformation of the gullet should be suspected. The obstruction to the passage of a soft catheter at about $4\frac{1}{2}$ inches from the gums, or X-ray examination immediately after attempting to swallow a teaspoonful or two of an opaque meal, will settle the diagnosis (Fig. 98).

The special type of deformity varies in different cases. The upper portion of the œsophagus, which is dilated, ends as a blind pouch a varying distance above the bifurcation of the trachea. The distal portion, which terminates at a slightly lower level, opens as a rule into the back of the trachea immediately above its bifurcation (Fig. 99) or, more rarely, into a bronchus. Sometimes, however, the distal portion does not communicate with the air passages, and at other times it is merely represented by a fibrous cord. On very rare occasions there is no interruption in the continuity of the œsophagus and the obstruction is of the nature of a membranous diaphragm (p. 330). As this last variety of deformity can be relieved by operation (incision of the membranous partition), it is advisable in all cases to practise a definite routine in order to discover the exact state of matters. At the earliest possible moment gastrostomy should be performed and an opaque medium (barium emulsion) introduced into the upper and lower portions of the œsophagus and an X-ray picture taken. In this way it is revealed whether or not the two portions of the gullet are in juxtaposition, and whether or not the lower portion opens into the air passages. Except in

the special type above mentioned no permanent relief is obtained by surgical intervention. The child may be kept alive for a longer period by feeding through a gastrostomy,

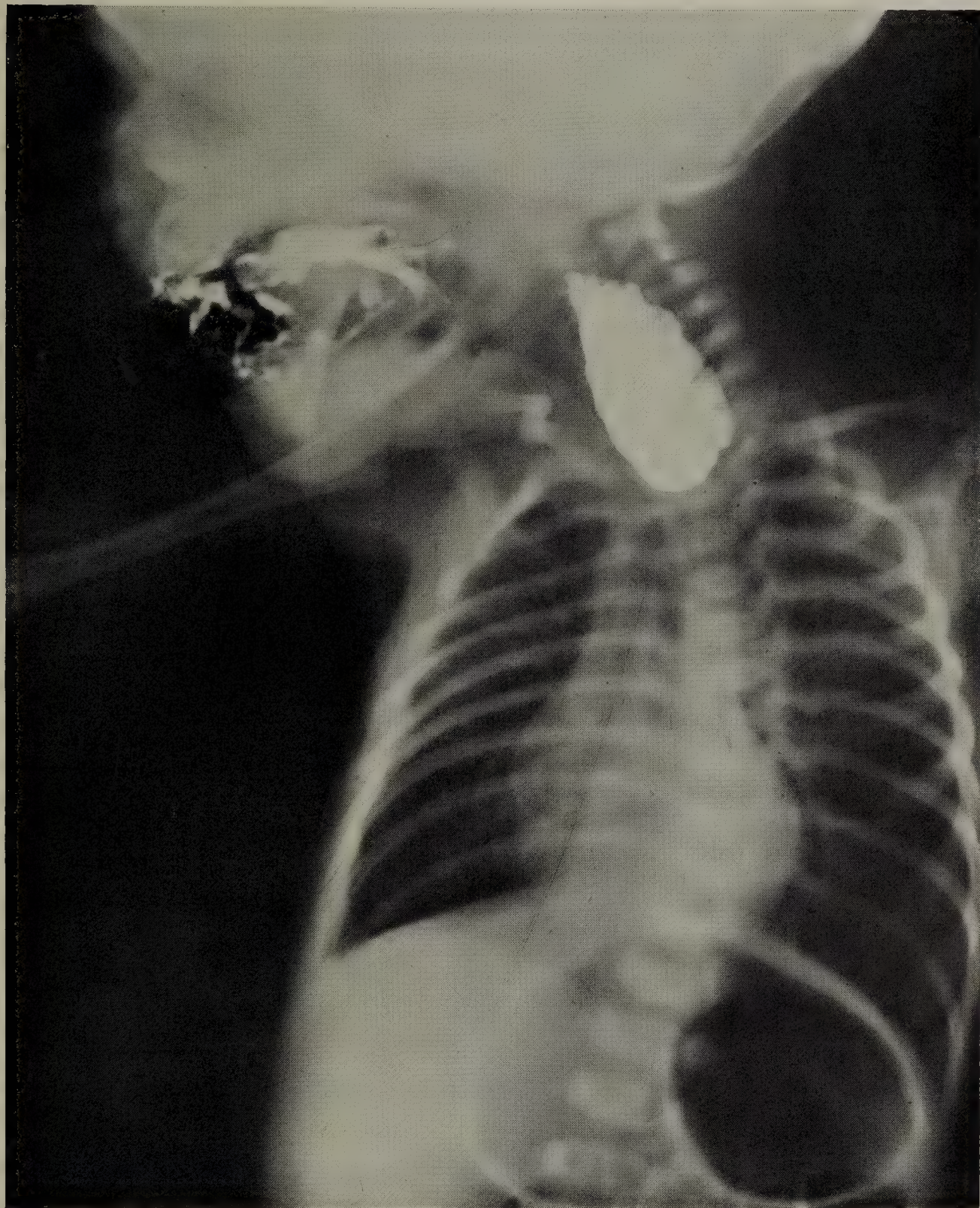


FIG. 98.—Skiagram after swallowing a few mouthfuls of an opaque meal, showing the dilated and obstructed upper end of œsophagus. (Boy aged 7 days.)

but the most frequent cause of death is not inanition but a pulmonary infection induced by the regurgitation of the stomach contents through the tracheal opening.

Congenital Stenosis.¹—Under this heading we intend to

¹ L. Findlay, *Acta Pædiatrica*, 1932, xiii, 70.



FIG. 99.—Congenital Atresia of Œsophagus.
(Girl aged $3\frac{1}{2}$ days.)

A probe (B) is inserted into upper dilated portion of Œsophagus which ends blindly. The lower portion of Œsophagus is seen to enter trachea (A) just above its bifurcation. (From a coloured drawing by Miss C. Brown Kelly of an example in R.H.S.C., Glasgow.)

include all examples of stenosis of the Œsophagus which do not follow injury, for the simple reason that it is at the present moment impossible to suggest a satisfactory classification. In some cases, although the symptoms do not appear till towards the end of the first year, or even later, the condition is undoubtedly congenital in origin. It is merely the degree of the stenosis and the change to a more solid type of food which have determined the late onset of the symptoms. On the other hand, there are cases in which the symptoms appear during the first weeks of life but which, because of the progressive nature of the difficulty, are probably not developmental in origin, but the absence of a sufficiency of satisfactory pathological material precludes a proper differentiation. Since, however, the symptomatology and the methods of investigation are the same in all types, it would appear expedient from this point of view alone that all examples should be grouped together under one heading.

Stenosis of the Œso-

phagus is almost certainly much commoner than atresia, although a study of the literature would create a different impression. It is only within recent years that the condition has become generally recognised. It is because it is rarely fatal, at least during childhood, that post-mortem records disclose its presence relatively seldom in comparison with the other and more fatal types of obstruction of the digestive tract.

Sex Incidence.—Boys are more frequently attacked than girls. Of 56 cases under thirteen years of age recorded in the literature or personally observed, and in which the sex is known, 39 were boys and 17 girls.

Seat of the Stenosis.—Although stenosis of the œsophagus may be present at any point in its course, there are two levels at which this is most prone to occur. These seats of election are (*a*) the level of the seventh and eighth dorsal vertebræ, and (*b*) the neighbourhood of the cardia. Of a series of 62 examples in children under thirteen years of age, either recorded in the literature or personally observed, 34 were situated about the level of the seventh dorsal vertebra and 19 in the neighbourhood of the cardia. Four occurred about the level of the fourth to fifth dorsal vertebræ, four at the level of the second dorsal vertebra, and one at the entrance to the gullet.

Nature of the Obstruction.—The nature of the obstruction varies and this does not seem to bear any relationship to the particular portion of the œsophagus affected. Simple narrowing of the canal without any apparent change in its wall, a membranous diaphragm, a localised fibrous or fibro-muscular thickening of the wall of the gullet and spasmodic contraction have been noted at the various levels. In the majority of recorded cases, however, the exact nature of the mischief remains in doubt. This is, as previously mentioned, due to the paucity of pathological material and to the fact that the most recent method of diagnosis (endoscopy) had not been employed. It is hoped that with a more general practice of endoscopy much exact information in this field will be forthcoming, and a satisfactory classification ultimately made possible.

(*a*) *Simple Narrowing of the Gullet.*—The gullet is normally somewhat narrower at the upper end, in the region of the bifurcation of the trachea, and at the cardia, and it is generally

held that it is at these levels where strictures of spastic origin or those resulting from injury by caustics are apt to occur. Narrowing in other regions and of a definitely pathological nature has, however, not infrequently been described.

For the most part this type of lesion has been noted in the adult, but this is probably because the condition, of itself, is not serious and does not shorten life. Kelly,¹ who recently collected from the literature all examples where the narrowing occurred at the upper end of the gullet, and at the same time records two personally observed, notes that in not a few of these dysphagia dated from early life. An undoubted congenital example is recorded by Cruveilhier² in a child who died at the age of two days. The upper part of the œsophagus was dilated with a narrowing of the lumen for a short distance about two inches above the cardia. Hirschsprung³ also records a similar type of lesion in a child of five months who had commenced to vomit shortly before: at the autopsy there was dilatation of the upper two-thirds of the œsophagus, then a very narrowed part, and then a normal part; the œsophageal wall at the seat of the stenosis was very thin, but above this level was definitely thickened.

Simple narrowing of the gullet is usually ascribed to a developmental defect such as occurs in the case of atresia. In view of the fact that atresia is practically constant in its situation, whereas narrowing may be found at any level, some other cause is probably at play. A more likely explanation is that the narrowing results from a failure of vacuolation of the œsophageal wall. In the process of development of the œsophagus, increase in the lumen is brought about by vacuolation of the wall; the vacuoles gradually enlarge and finally rupture into the lumen, thus bringing about an increase in its size.

(b) *Membranous Diaphragm*.—This type of obstruction has been described by Vinsen⁴ in the upper part of the œsophagus on a level with the second and third dorsal vertebræ and also in the region of the cardia. Beatty,⁵ Heatly,⁶ and Kelly and

¹ A. B. Kelly, *Journ. Laryng. and Otology*, 1931, xlv., 521.

² J. Cruveilhier, *Anatomie Pathologique*, Paris, 1835, T. I, L. xxxviii.

³ Hirschsprung, *Hosp. Tid.*, 4 R., Bd. iii., 1861.

⁴ P. P. Vinsen, *Collected Papers of Mayo Clinic*, 1923, xv., 3.

⁵ C. C. Beatty, *Brit. Journ. Child. Dis.*, 1928, xxv., 237.

⁶ C. A. Heatly, *Arch. Otolaryngology*, 1928, viii., 66.

Findlay¹ have all described an example of a membranous obstruction about the level of the seventh dorsal vertebra. In the cases described by Heatly, and Kelly and Findlay, X-ray examination showed dilatation of the portion of the gullet below as well as above the obstruction.

The cause of this type of obstruction is probably a perversion of the developmental vacuolation which has been mentioned as a possible etiological factor in narrowing. It seems feasible that a portion of the wall of one of the vacuoles should persist in the form of a valve.

(c) *Fibrous or Fibro-muscular Thickening of Wall of Œsophagus*.—Hutchison² has recorded the case of a boy of three years who had been subject to periodic attacks of vomiting since the age of six weeks, and in whom X-ray examination after a barium meal showed a constriction at the level of the seventh dorsal vertebra. Soon after coming under observation he developed a fatal attack of broncho-pneumonia, and at the autopsy a "hard fibrous constriction of the Œsophagus was found at a level of $1\frac{1}{2}$ inches below the bifurcation of the trachea." Ashby³ has described a somewhat similar lesion at the lower end of the Œsophagus, and one of us (L. F.) has observed a like type of obstruction in a child of five months. This patient had no trouble till the age of eight weeks, when regurgitation of food commenced and gradually increased in severity till at the age of five months no food could be swallowed. Death resulted from inanition and at the post-mortem examination a fibrous thickening of the whole circumference of the Œsophagus was discovered at a level of the junction of first and second dorsal vertebræ, and causing such extreme stenosis that a probe was only passed with difficulty.

It is possible that the same pathological process is not at work in all these cases. Histological examination of Ashby's case revealed hypertrophy of muscle with fibrosis, and in the case observed by one of us (L. F.) fibrosis. In Hutchison's case no microscopic examination of the thickened part of the Œsophagus was carried out. Ashby considered the condition analogous to hypertrophic pyloric stenosis, as did also Strauss

¹ L. Findlay and A. B. Kelly, *Proc. Roy. Soc. Med.* (Sect. Laryng.), 1931, xxiv., 85.

² R. Hutchison, *Proc. Roy. Soc. Med.*, 1922, xvi. (Sect. Dis. Child., p. 42).

³ H. T. Ashby, *Proc. Roy. Soc. Med.*, 1920, xiii. (Sect. Dis. Child., 146).

and Hess,¹ and, since the lesion in their patients was situated at the level of the cardia, this seemed quite a probable explanation; but the situation of the lesion in Hutchison's case (seventh dorsal vertebra), as well as in our own (first to second dorsal vertebræ), and their progressive nature, with the additional fact that the pathological process was in the two examples histologically examined wholly or in part fibrous, render such an analogy untenable. In none of the cases was there any evidence of syphilis.

(d) *Short Œsophagus*.—From endoscopic examination in some cases in which the symptoms dated from birth, and in which the stenosis was situated at the level of the seventh dorsal vertebra, Kelly and Findlay² concluded that the condition was of the nature of a congenitally short Œsophagus, and that the constricted portion was the abnormally situated cardia. In these particular examples not only did the stenosis, as seen by the endoscope, suggest the cardia, but X-ray examination after a barium meal revealed a dilatation of the alimentary tract below the constriction which simulated a partial hiatal hernia (Fig. 100), and from the lining of which gastric mucosa was recovered. This would seem conclusive proof that the state of matters was due to a short Œsophagus, but one hesitates to be dogmatic in the absence of pathological evidence, more especially since in the normal child almost similar radiological appearances are not infrequently observed (Fig. 101), and gastric mucosa can be recovered from the lining of the alimentary canal well above the level of the diaphragm. It would appear that in childhood hiatal hernia, at least during swallowing, is a physiological process. It should also be pointed out that the same appearance of hiatal hernia was observed in the above quoted examples of membranous obstruction situated at the level of the seventh dorsal vertebra reported by Heatly and by Kelly and Findlay (p. 330).

(e) *Spasm of the Œsophagus*.—Although spastic conditions of the Œsophagus are rarer in the child than in the adult, undoubted instances are on record. Sheldon and Ogilvie³ report

¹ A. A. Strauss and J. H. Hess, *Journ. Amer. Med. Assoc.*, 1925, lxxxiv., 501.

² L. Findlay and A. B. Kelly, *Proc. Roy. Soc. Med. (Sect. Laryng.)*, 1931, xxiv., 85.

³ W. Sheldon and A. G. Ogilvie, *Arch. Dis. Child.*, 1929, iv., 347.

the case of a girl who had vomited from birth, and when seen at the age of two and a half years presented the typical radiological signs of œsophageal stenosis at the level of the seventh dorsal vertebra; at the age of four years all vomiting ceased; at five years she succumbed to an intercurrent attack of

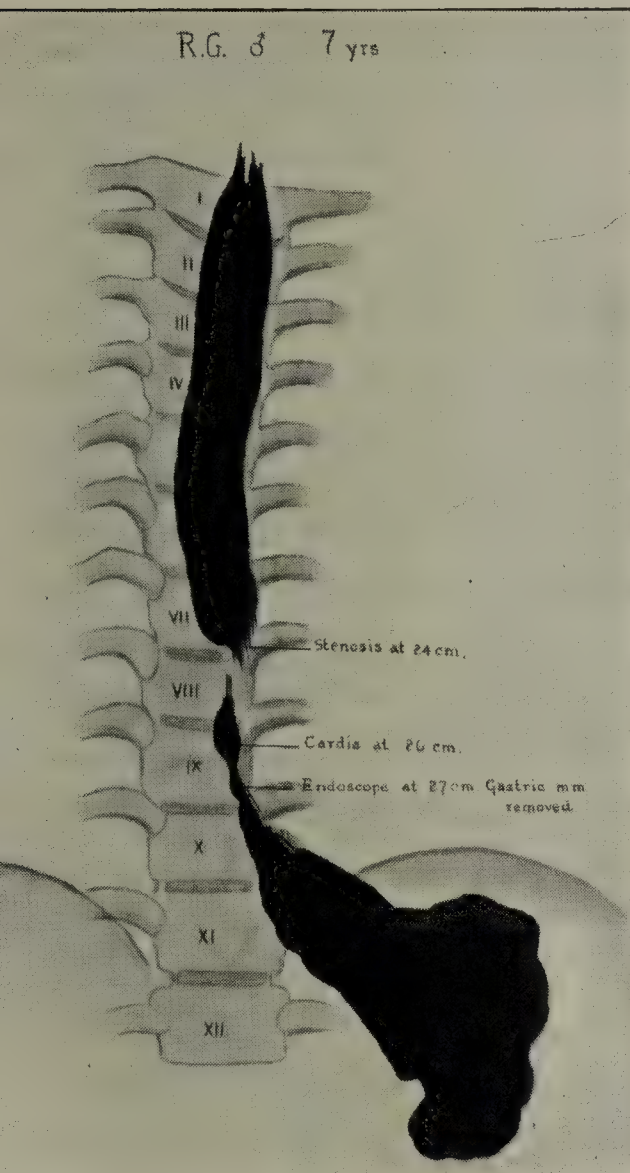


FIG. 100.—Skiagram in case of Œsophageal Stenosis in a child of 7 years. Note appearance of hiatal hernia. Gastric mucosa was obtained from the lumen at a level of 9th dorsal vertebra. (From a drawing by Miss C. B. Kelly.)

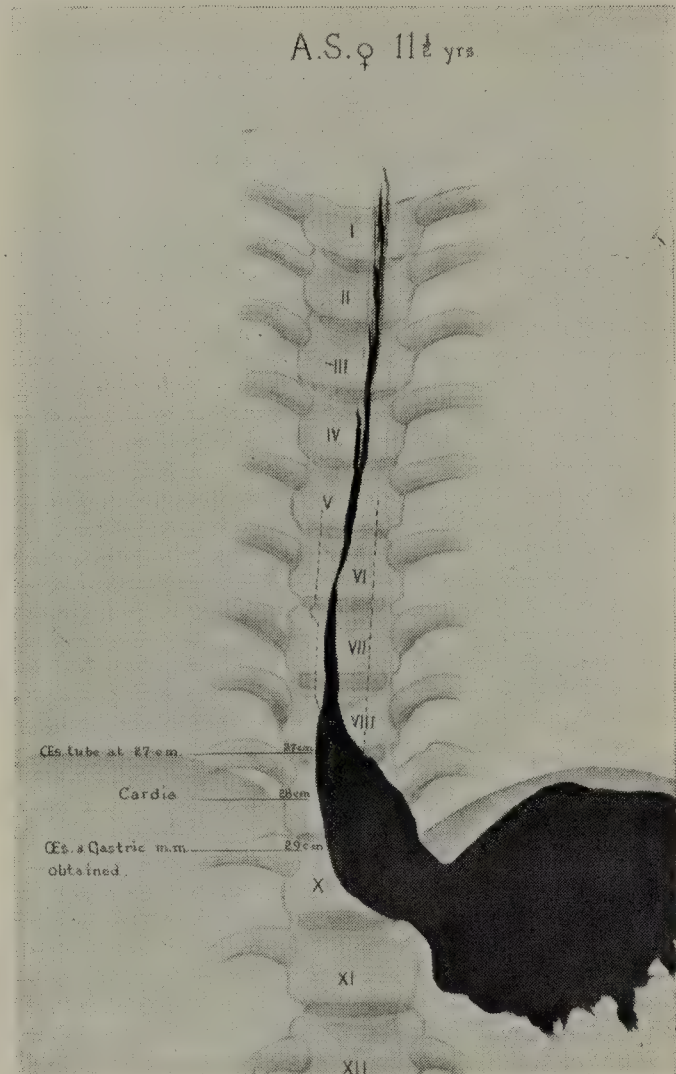


FIG. 101.—Skiagram taken during swallowing a pultaceous opaque bolus in a child aged 11½ years without Stenosis of Œsophagus. Note appearance of hiatal hernia. Gastric mucosa was obtained from the lumen at a level of junction of 9th and 10th dorsal vertebræ. (From a drawing by Miss C. B. Kelly.)

tuberculous meningitis, when the autopsy disclosed absolutely nothing abnormal in the œsophagus. A further example observed by Peugniez¹ affected a much emaciated boy of seven years who had always experienced dysphagia, and in whom X-ray examination revealed a stenosis at the level of the junction of the fourth and fifth dorsal vertebræ. Bougies were employed, and for three days the boy became hysterical on each

¹ M. Peugniez, *Bull. et Mem. de la Soc. de Chir. de Paris*, 1928, xx., 158.

occasion when the instruments were passed and offered the greatest resistance, but on the third day he said that he did not require to be held, and from that time bougies could be passed easily and all dysphagia disappeared. Monrad¹ also records the case of a boy of five years who from birth had vomited, or rather regurgitated, his food during the whole meal. When

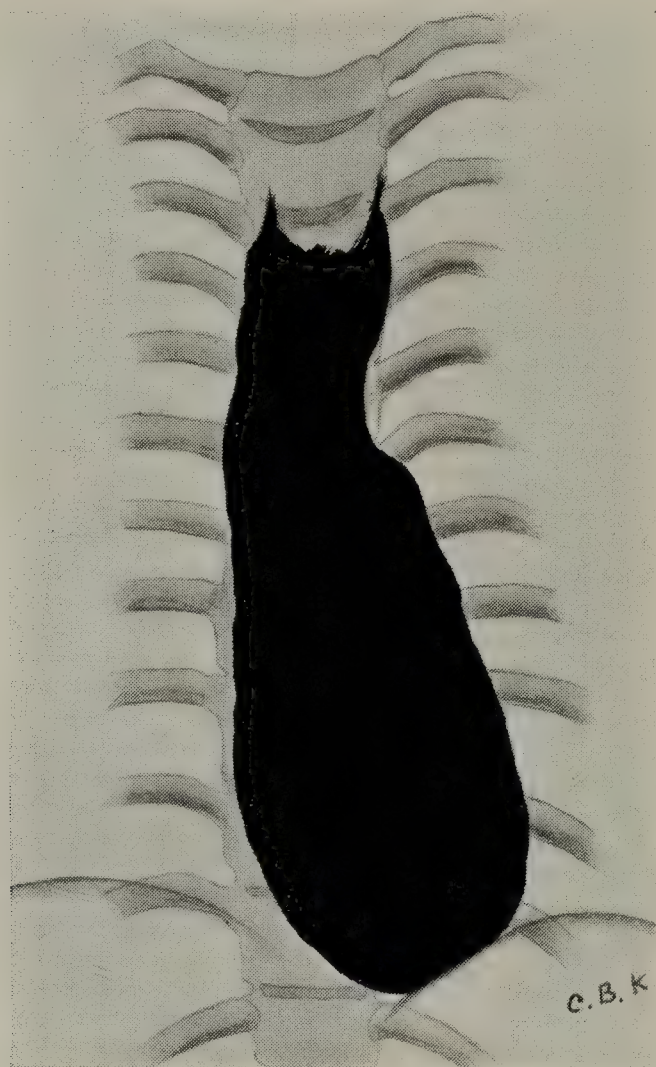


FIG. 102.—Tracing from X-ray photograph after a barium meal in an example of Cardiospasm (boy aged $3\frac{3}{4}$ years). Note great distension of œsophagus throughout its whole length. (From a drawing made by Miss C. B. Kelly.)

first seen an obstruction was detected at a level of 25 cm. from the teeth. The passage of a bougie caused for a time a complete disappearance of the symptoms, but the dysphagia relapsed some time later, when the obstruction was found to be situated only 15 to 18 cm. from the teeth, and on this occasion was clearly shown by X-ray examination on a level with the sixth dorsal vertebra. Again the passage of a bougie caused an immediate, but this time a permanent, disappearance of the symptoms.

(f) *Cardiospasm* (so-called *Idiopathic Dilatation of the Œsophagus or Achalasia of the Cardia*).—Although this is an affection which is not generally considered congenital in origin, apparent

examples are on record (Castronovo,² Cameron³) in which the symptoms had been present from birth or early infancy. In cardiospasm there is dilatation of the whole length of the œsophagus, but especially of the lower two-thirds (Fig. 102), with a varying degree of hypertrophy of the wall. It would obviously seem secondary to an obstruction at

¹ S. Monrad, *Acta Pædiatrica*, 1921, i., 29.

² E. Castronovo, *La Pediatria*, 1925, xxxiii., 483.

³ J. A. M. Cameron, *Arch. Dis. Child.*, 1927, ii., 358.

the lower end of the gullet, although post-mortem nothing abnormal is detected.

Mikulicz's hypothesis that there is a spasmodic contraction of the cardia is probably correct, and this may be due, as Kelly¹ has pointed out, to some disturbance of the intrinsic nervous system of the Œsophagus, although it may also be in response to dysfunction of the vagus or Auerbach's plexus. H. D. Rolleston² suggested that the obstruction is due to failure of the co-ordinating mechanism by which the cardia is relaxed during swallowing, and that paralysis or continued inhibition of the longitudinal muscular fibres of the Œsophagus would allow dilatation of the tube to occur, and, at the same time, by interfering with the opening of the cardia, would induce hypertrophy of the circular muscular coat. Hurst³ also is of opinion that the condition is one of inco-ordination, for which he originated the term "achalasia." This theory of etiology, however, imbues the tone of involuntary muscle with a power which it does not possess, as often definite force is required to overcome the obstruction. In any case, by endoscopic examination Kelly has seen contraction and relaxation of the cardia occur.

Symptomatology.—Whatever the nature of the lesion the symptomatology is more or less alike. On the degree of stenosis depend the age of onset of the symptoms, as also their severity. In many cases difficulty with swallowing appears soon after birth, but in other cases not until the time of weaning, when the fluid milk-diet is changed for one of a more solid nature. In some cases the trouble with feeding is present at birth, but soon disappears, and only reappears on weaning, or even later, and in still other cases, especially those of cardio-spasm, the symptoms may not make their appearance till later childhood.

The characteristic symptom of Œsophageal stenosis is the regurgitation of food during a meal, although advice will have been sought for what the parents call vomiting. Vomiting, or what appears to be vomiting, after a meal does occur, but it is the regurgitation of food during the whole meal which is the special feature, and this should always suggest the passage of a soft rubber-tube for the detection of Œsophageal stenosis.

¹ A. B. Kelly, *Brit. Med. Journ.*, 1912, ii., 1047.

² H. D. Rolleston, *Trans. Path. Soc. London*, 1896, xlvii., 37.

³ A. F. Hurst, *Quart. Journ. Med.*, 1914-15, viii., 300.

Dysphagia is seldom complained of by these children, but if they be observed during a meal it will be noticed that they drink very slowly, take very small mouthfuls, and chew any solid food very thoroughly before swallowing. For example, a child may take an hour to consume a meal consisting of a little porridge, a slice of bread, and some cocoa.

The symptoms do not necessarily occur after every meal. They may intermit for a day or more at a time. They are always more marked if the child is ill from any other cause, and that irrespective of whether the stenosis is apparently organic or spastic in nature. In view of the fact that much of the food is lost, these patients are usually constipated and suffer in nutrition so that they are much under height and weight.

Diagnosis.—As previously mentioned, regurgitation of food during a meal, especially if this has been present from birth, should always suggest œsophageal obstruction and the passage of a soft rubber-tube for its detection. If an obstruction be present this will almost certainly be detected by this means, and not only will the seat of the lesion but also the degree of stenosis be discovered in this way. In some cases the tube cannot be introduced into the stomach, no matter how much force is employed, but in others, when the degree of stenosis is not so marked, after a little pressure the resistance will be overcome and the tube will enter the stomach. X-ray examination after a barium meal, however, is the surest means by which the presence and situation of an obstruction can be detected, but it must be borne in mind that unless the emulsion employed is of a thick consistence, like porridge, and the examination is made at once, time may be given for the opaque substance to pass through the narrowed part and nothing abnormal will be observed. For the diagnosis of the particular type of obstruction (narrowing, spasm, or membranous diaphragm), and the decision of the appropriate treatment, endoscopic examination is essential and should never be omitted. The radiographic pictures of all types of stenosis are identical.

Treatment.—This, of course, depends on the character of the lesion. Incision of a membranous obstruction may give immediate and permanent relief. For the other types dilatation by gum-elastic bougies (which older children can be taught to do for themselves) may be tried, but the results are

often disappointing. It is comforting to remember that the condition in some instances, probably when of a spastic nature, undergoes spontaneous cure.

Diverticula of Œsophagus.—Congenital diverticula of the Œsophagus may closely simulate the clinical picture of Œsophageal stenosis. One of us (L. F.) observed an example of a long sausage-shaped diverticulum situated towards the lower end and which gave rise to persistent vomiting during the first year of life. Thereafter all vomiting ceased and the child developed normally, although the diverticulum was quite apparent by X-ray examination at the age of eleven years.

It may be remarked that other types of diverticula (pulsion and traction diverticula) are unknown during childhood. This is somewhat remarkable as it would seem certain that the cause of the former lies in some local weakness of the Œsophageal wall, and especially when one recollects how frequent is chronic inflammatory glandular mischief in the mediastinum during the earlier years of life.

Congenital Hypertrophy of the Pylorus

(Hypertrophic Pyloric Stenosis).

Three decades ago congenital hypertrophy of the pylorus was regarded as a pathological curiosity only diagnosed after death, and always fatal. Now the disease is looked upon as one which any general practitioner may expect to meet with, and which he ought to be able to recognise. It has also passed out of the ranks of incurable disorders, and, in the large majority of cases, can be treated with complete and permanent success.

Symptoms.—In a typical case of pyloric hypertrophy the symptoms are very characteristic. The patient is a baby of a few weeks old, a boy in four cases out of every five, and most frequently the first born, and he comes of normal or sometimes of distinctly nervous parents. He has usually been born at the full-time, after a normal pregnancy and a normal labour.

Thomson knew of at least four instances in which two children in a family were affected, one in which the patient's father had suffered from similar symptoms in early infancy, and one in which the mother's brother had died of the disease. The present editor has known of the third, fourth, and fifth

children in a family so affected, and he has recently heard of an instance in which twins were both the subject of the malady.

The complaints with which the child is brought are usually the very ordinary ones of *vomiting* and *wasting*, but it will be noted that there has rarely been any dietetic error sufficient to account for the onset of the trouble. Most of the patients have been either on the breast at the time, or else carefully fed on the bottle. If changes have been made in the character of the food, after the vomiting began, it will generally have been noticed that no improvement lasted for more than a day or two. The only change, if any, which has done good for longer will probably have been in the direction of lessening the bulk of the fluid taken. Occasionally some vomiting is said to have existed since birth; but usually it has only become severe after two or three weeks or more of apparently perfect health. An attack of hæmatemesis may be the first symptom. The character of the vomiting, when it has lasted for any time, is in most cases peculiar. It is forcible and explosive; and the mother says that the milk shoots out on to the floor through the mouth and nose. The vomited matter is often large in amount, representing more than one meal. It is to be noticed also that, apart from the vomiting, there have usually been very few of the ordinary signs of dyspepsia. The tongue has kept clean, and there have been no sour eructations, no flatulent distension of the bowels, usually no bile in the vomit, and no diarrhœa. When the vomiting is severe, the motions are noticeably small and usually dark in colour (hunger stool), and the urine scanty.

As the vomiting continues there is generally a steady loss of weight, and the amount of this, along with that of the fæcal matter passed, are of great importance, because they indicate the degree to which the narrowed pylorus is preventing the passage of food into the bowel, and consequently the degree of urgency of the case. Although the child may be wasted he, in the early stages, will not appear prostrate or toxic, as a child with dyspepsia, and the elasticity of the tissues may remain unimpaired. He usually loses colour and is pale, and in the later stages of the disease he becomes definitely drowsy, with slow shallow breathing and long periods of apnœa due to an accompanying alkalosis.

The abdomen in cases which have lasted for any time is

sometimes spoken of as pear-shaped. Over the stomach region it is distended, and in cases of many weeks' duration the recti are hypertrophied and show distinct separation there; while the lower part of the abdomen is small and normal in appearance.

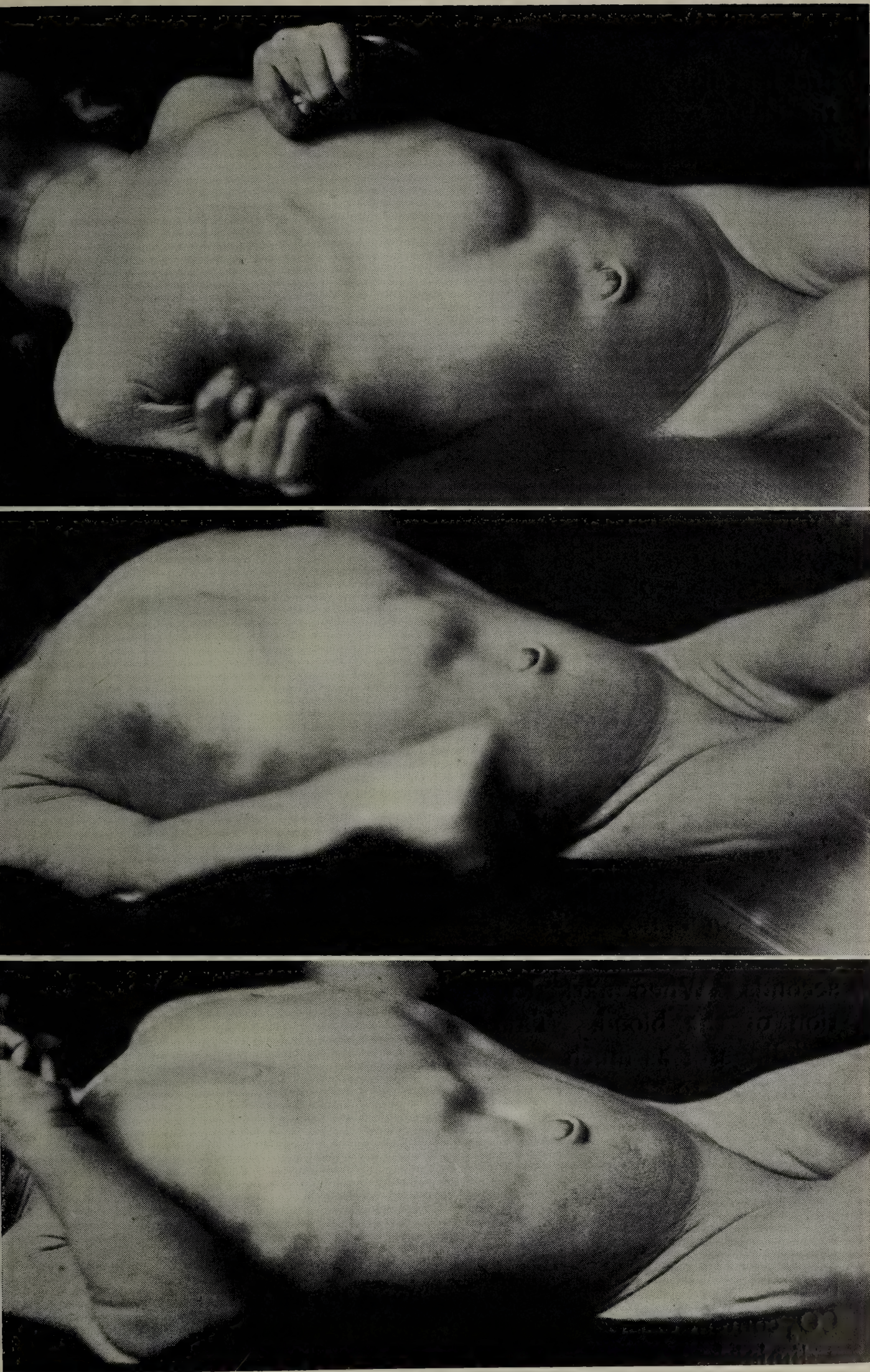
The above group of symptoms in a baby under three or four months old should always make one suspect pyloric stenosis, and demand a search for the pathognomonic features of the disease. These are a *palpable tumour* and *marked visible gastric peristalsis*.

The examination is best carried out with the baby lying on the lap of the mother or nurse, seated in front of a warm fire. The physician, sitting on the child's left side and facing him, applies the left hand to the right hypochondrium and by gradually increasing the pressure explores the abdomen more and more deeply. The *pyloric tumour* has the size of the terminal phalynx of the finger, varies somewhat from time to time on account of muscular contractions, is situated as a rule slightly above and to the right of the umbilicus, although it may be high up under the edge of the liver or low down and to the right in the iliac fossa, and is usually more or less fixed, although it may be movable. Sometimes great difficulty is experienced in detecting the tumour. It would seem at times as if a distended stomach may obscure it, whereas in other cases it is more readily palpable when the stomach is full. For these reasons it is wise to search for the pylorus with the stomach full, *i.e.*, after a feed, and also when the viscus is empty, *i.e.*, after a large vomit or the passage of the stomach-tube. With care and patience, however, and attention to the above points, a pyloric tumour can be felt in practically every case, if not on the first occasion then on the second, and that quite irrespective of the duration of the symptoms.

The other characteristic feature of the condition is *visible exaggerated gastric peristalsis* as represented in Figs. 103 to 108. The outline of the stomach stands out and forcible movements pass over it from left to right. In its most marked degree, a rounded swelling, sometimes almost the size of a golf-ball, rises up slowly in the left hypochondrium and rolls to the right across the abdomen. By the time it reaches the umbilicus another is seen following it; and, if the stomach is much dilated, a third may be showing under the left costal margin before the



EXAGGERATED GASTRIC PERISTALSIS IN CONGENITAL PYLORIC HYPERTROPHY.
FIGS. 103 to 105.—Boy of 8 weeks ; Gastro-enterostomy, by Sir Harold Stiles ; recovery.
(Photographs by Dr T. B. Hamilton.)



EXAGGERATED GASTRIC PERISTALSIS IN CONGENITAL PYLORIC HYPERTROPHY.
 FIGS. 106 to 108.—Boy of 8 weeks ; Gastro-enterostomy, by Sir Harold Stiles ; recovery.
 (Photographs by Dr T. B. Hamilton.)

first has disappeared. While this visible peristalsis is going on, the child may show signs of cramp-like pain; but, generally, there is no pain at all. It is important to bear in mind that there may be no peristaltic movements of the stomach if it is empty, and hence before deciding that it is not present the child must be given a feed. If the child is not comfortable and wriggles about, contractions of the muscles of the abdominal wall will obscure it. The abdominal wall should be warm, and gentle friction or tapping over the stomach often help to set it up. It must also be remembered that in order to be diagnostic the peristalsis must be very marked. In any child, when the stomach is full it stands out, the outlines of which vary in prominence from time to time, and in the very much emaciated child very slight peristaltic waves may be seen. The peristalsis of pyloric stenosis not only reveals an unmistakable dilatation of the viscus but also a hypertrophy of its wall.

Another important and almost pathognomonic feature of pyloric stenosis is the state of *alkalosis* above mentioned. So common is this in pyloric stenosis, and so rarely is it found in any other disease, that we have had our attention directed to the presence of pyloric stenosis, although the child had never vomited and the bowels were moving regularly, by recognising the existence of alkalosis. In the alkalotic state the child is unduly drowsy, pale, with slow shallow breathing and long periods of apnœa. The respirations may only number eight per minute and the periods of apnœa last for as long as thirty seconds. When marked it will usually be verified by examination of the blood. This will reveal a high CO_2 content. Readings of as much as 148 vols. CO_2 per cent. instead of the normal 45 to 55 vols. per cent. have been observed. The blood invariably shows at the same time a lowered content of the acid radicle chlorine, and it is this which is usually considered the cause of the alkalotic state. That this is, however, not the whole explanation is revealed by the facts that the fall in chlorine and the rise in CO_2 are not proportionate, and that the blood chlorine can be rendered normal without influencing the CO_2 content or the rate of the breathing. The tissues also show a diminished chlorine content. The fall in the chlorine of the blood and tissues is generally ascribed to the loss of chlorine in the vomit, but the development of alkalosis in the absence of

all vomiting, as noted in the case above quoted, and the absence of alkalosis in other types of vomiting (gastro-enteritis and tubercular meningitis) render such an explanation not completely satisfactory. Morris¹ has drawn attention to the fact that the chlorine in the body is present in a volatile as well as in a non-volatile form, and has suggested that since it is only the non-volatile form which is regularly estimated, the diminution may be due to a change in the proportion of these two forms, and thus more apparent than real. It may be that the chlorine is deflected to detoxicate some of the products of protein break-down.

It will usually be found in pyloric stenosis that the urinary chlorides are very scanty, and, as Morris and Graham² have shown, that 80 to 90 per cent. of any chlorine injected intravenously is retained, whereas in health 80 to 90 per cent. is excreted.

In spite of the frequency of the accompanying alkalosis tetany is very rare. Convulsions with a facial phenomenon are usually late and terminal features.

The severity of the symptoms varies greatly in different patients. There is, for example, a severe type of case in which the vomiting sets in during the first days of life, is urgent from the beginning, and rapidly gets worse. There is another type of case in which, despite the palpable tumour and very exaggerated peristalsis of a much dilated stomach, the vomiting is not particularly severe and the fæcal matter is fairly abundant; this variety of case is very amenable to medical treatment. Between these two extremes all gradations are encountered.

On pathological examination there is not very much difference to be found in the state of the stomach or degree of pyloric hypertrophy in these different types. It is quite evidently the functional rather than the anatomical abnormality which determines the severity of the symptoms.

In cases in which the pyloric closure is not relieved by the treatment, the muscular strength of the stomach fails after a time. The child is then no longer able to relieve himself sufficiently by vomiting, and, if the stomach-tube is not regularly used, the stomach becomes greatly over-distended and severe

¹ N. Morris and S. Morris, *Biochem. Journ.*, 1930, xxiv., 1716.

² N. Morris and S. Graham, *Arch. Dis. Child.*, 1931, vi., 27

catarrh sets in. The child's resistance becomes lowered and he is liable to develop some intercurrent infection, *e.g.*, bronchopneumonia, pyelonephritis, and gastro-enteritis. This last complication is extremely dangerous, and often ends fatally in cases which had seemed to be recovering satisfactorily. Contrary to what might have been expected the presence of alkalosis is of little special gravity. In degree it varies as a rule with the general state of the child's nutrition, and thus is more a measure of the severity of the inanition than evidence of any particular complication.

Diagnosis.—It can be confidently stated that no disease of infancy can be more frequently correctly diagnosed, so long as one is alive to the possibility of its presence. In practically every instance the pyloric tumour can be detected, and the gastric peristalsis can be elicited, by anyone of experience, provided the directions described above are followed. For the diagnosis of pyloric stenosis these signs are not only pathognomonic but essential.

It is frequently stated that some time is necessary for the development of the visible peristalsis, but one of us (L. F.) has never seen a case too early after the onset of the symptoms for its detection. It may be present even in the absence of all symptoms (p. 342), and frequently it can be observed on the first day on which vomiting is recorded. In support of this statement it may be remarked that the condition has never been an accidental finding in the post-mortem room in any child in whom the possibility of pyloric stenosis had been entertained. On the other hand, many cases are on record in which neither tumour nor peristalsis were detected, and in which operation failed to reveal any pyloric hypertrophy. It cannot be too emphatically stated that vomiting *per se*, even when this is projectile and most inveterate, is no criterion of hypertrophic pyloric stenosis. Spasm of the pylorus there may be, but if there is hypertrophy then a tumour and gastric peristalsis will be apparent, if not at the first examination then on the occasion of the second. It is not so much a matter of time allowing for the development of the physical signs, as of the local conditions (general abdominal distension, contraction of the abdominal muscles, and the fullness of the stomach) influencing peristalsis and obscuring or bringing the pylorus more readily within the reach of the examining hand.

The presence of alkalosis as evidenced clinically by the slow shallow breathing, or on chemical examination of the blood by its increased CO_2 content, should always make one suspect pyloric stenosis, as it is found in practically no other infantile disease. Very occasionally it has been described in gastro-enteritis. Although diminution of the chlorides in the urine is common in pyloric stenosis it may occur in vomiting from whatever cause, and hence is of no diagnostic value. A normal chloride content of the urine, on the other hand, is distinctly against the presence of pyloric obstruction.

X-ray examination after a barium meal is sometimes recommended as a valuable help in diagnosis. In our opinion it is never necessary and is, moreover, not without danger. In a series of cases of hypertrophic pyloric stenosis occurring in The Infants Hospital, Vincent Square, Maizels¹ reports that "radiography was negative in three cases, and inconclusive in eleven others where the diagnosis was subsequently confirmed."

The cases most difficult to distinguish from pyloric stenosis are those of an obscure temporary nervous affection of the stomach which may be called "**pyloric spasm.**" Mild instances of this condition are frequently met with, and give little trouble either in the diagnosis or treatment. Severe cases may cause great difficulty, but they are uncommon. They are not only difficult to recognise at first but also very resistant to treatment, although they nearly all recover completely in time.

The patient is *usually a girl*, and there is generally much more evidence of pain than in cases of pyloric hypertrophy. The vomiting occurs shortly after each feed and the whole contents of the stomach are returned at once, or else it recurs at short intervals until the stomach is emptied. There is little or no tendency for the food to accumulate in the stomach, and there are no other signs of gastric dilatation or hypertrophy. Before the vomiting the outline of the stomach may stand out distinctly, but there is never any characteristic peristalsis to be seen. The motions and urine are scanty, and sometimes slight diarrhoea occurs.

Cases of pyloric spasm require the greatest care and perseverance in their treatment. The diet must, of course, be regulated and the stomach should be washed out regularly, if this is found to have any good effect on the vomiting. Often,

¹ M. Maizels, *Arch. Dis. Child.*, 1931, vi., 293.

however, these measures have disappointingly little influence on the habitual return of the meals. The treatment which has been found most useful is the cautious administration of small doses of laudanum, beginning with $\frac{1}{40}$ minim, and gradually increasing to $\frac{1}{10}$, or even to $\frac{1}{5}$ minim. When this succeeds in lessening the vomiting, the weight steadily rises. These cases, if the mother is careful and trustworthy, do better at home than in hospital, and home treatment is also preferable for them, because, like cases of pyloric hypertrophy, they are apt to go downhill rapidly if they are infected with diarrhœa. On post-mortem examination, the pylorus and stomach are found to be quite normal.

Congenital atresia or stenosis of the duodenum (p. 354) is sometimes mistaken for pyloric hypertrophy on account of the vomiting and visible peristalsis. In this condition the vomiting begins within the first few hours of life, the vomitus is frequently bile-stained, which practically never occurs in pyloric stenosis, the peristalsis is seldom so marked, and there is no pyloric tumour. Radiography is of the greatest help in the recognition of these deformities and should be carried out in every suspected case, as early operation gives a great measure of success.

Etiology.—The question of the origin of the muscular hypertrophy in this disease and its relation to the pyloric obstruction are still matters of dispute.

On the one hand, there are those who hold with Hirschsprung¹ and Cautley that the condition is a primary congenital hyperplasia, and that the abnormalities of its function are the result and not the cause of the muscular enlargement. The facts that a hypertrophied pylorus has been observed in the foetus and that the size of the pyloric tumour varies very little, if at all, with the duration of the symptoms or age of the infant lend strong support to this view.

On the other hand, there are many who agree with John Thomson,² who maintained that a true muscular hypertrophy of this sort *never* occurs as a primary phenomenon, but is always the result of antecedent overaction; and who therefore hold that, to account for the presence of such hypertrophy as is found in these cases, it must be assumed that some cause of habitual overwork—

¹ *Jahrb. f. Kinderheilk.*, 1888, xxviii., 61.

² *Brit. Med. Journ.*, 1895, ii., 711; *Edin. Hosp. Rep.*, 1896, iv., 116; *Scot. Med. and Surg. Journ.*, 1897, i., 511; *Brit. Med. Journ.*, 1902, ii., 678.

such as inco-ordination between the stomach and the pylorus—has existed for a long time previously. This state of matters has been likened to that occurring in cardiospasm or achalasia of the cardia. The conditions are, however, not strictly comparable, as in the one hypertrophy is the rule and in the other is conspicuous by its absence. In this connection it is interesting to record that Gaisford¹ has reported the apparent development of a second pyloric tumour proximal to the first which had previously been operated upon.

Morbid Anatomy.—The condition found on post-mortem examination is a very simple one, and it is interesting to observe how it corresponds with the symptoms.

The essential *structural* change is confined to the upper portion of the alimentary tract. It consists in a high degree of pure hypertrophy of the muscular coat of the pylorus and adjacent stomach wall, and a much less amount of the same thing in the œsophagus. The other changes present, such as dilatation and catarrh of the cardiac end of the stomach, and the general marasmus, are obviously secondary results of the pyloric hypertrophy and the obstruction which it causes.

The tumour of the pylorus which the muscular hypertrophy occasions is very striking. It forms a hard, tense, elastic swelling, bulging in the centre (Figs. 109 to 111). The peritoneal coat is distended to its utmost limit and so tightly stretched by the increased bulk of the muscle within it that the pylorus is circular on transverse section. The inability of the outer coats to stretch further is a point of practical importance, for it results in the mucous and submucous coats becoming increasingly compressed as the muscular layer thickens and thus constitutes an active element in the obstruction of the pyloric lumen. The apparent narrowing of the mucous



FIG. 109. — Stomach with Congenital Hypertrophy of Pylorus, showing the thickening of muscle in pylorus and prepyloric portion.

¹ W. Gaisford, *Arch. Dis. Child.*, 1931, vi., 111.

coat as seen on section may be due partly to this compression and partly to elongation caused by the contractions of the hypertrophied muscle. The apparent thickening of the submucous coat and the folds in the mucous membrane also probably result from the compression exerted by the surrounding muscular tissue.

The essential abnormality of *function* is as strictly localised as that of the structure. It consists in an ill-timed, abnormally forcible, and prolonged closure of the pylorus. That spasm of the hypertrophied muscle is the chief determining factor in the



FIG. 110.—Transverse Section of Normal Pylorus, near the Duodenum, $\times 4$ diam. (From a child of 9 weeks.)



FIG. 111.—Transverse Section of Hypertrophied Pylorus, near the Duodenum, $\times 4$ diam. (From a child of 9 weeks.)

condition can only explain the sudden onset in many of the cases and the intermission of all symptoms for two or three days at a time, as well as their not infrequent sudden cessation. Nevertheless, mechanical blocking by the bulk of the muscle and the rugose and perhaps œdematous mucosa are possibly contributory factors.

Treatment.—That pyloric stenosis may recover of its own accord, or that medical measures not infrequently result in complete cure, no one who has any extensive experience of the condition can doubt. One of us (L. F.) has seen on several occasions an emaciated infant shortly after the cessation of severe and inveterate vomiting, which had been present for many weeks, and in whom physical examination revealed the

presence of a pyloric tumour and visible gastric peristalsis. The true nature of the malady had never been diagnosed and no treatment other than ringing the changes on the type of food had been adopted. The spasm which we have said to be the direct cause of the symptoms tends to disappear during the fourth month and then, if the child lives long enough, a natural cure results. The accompanying Chart (Fig. 112) shows the course of events in a child medically treated.

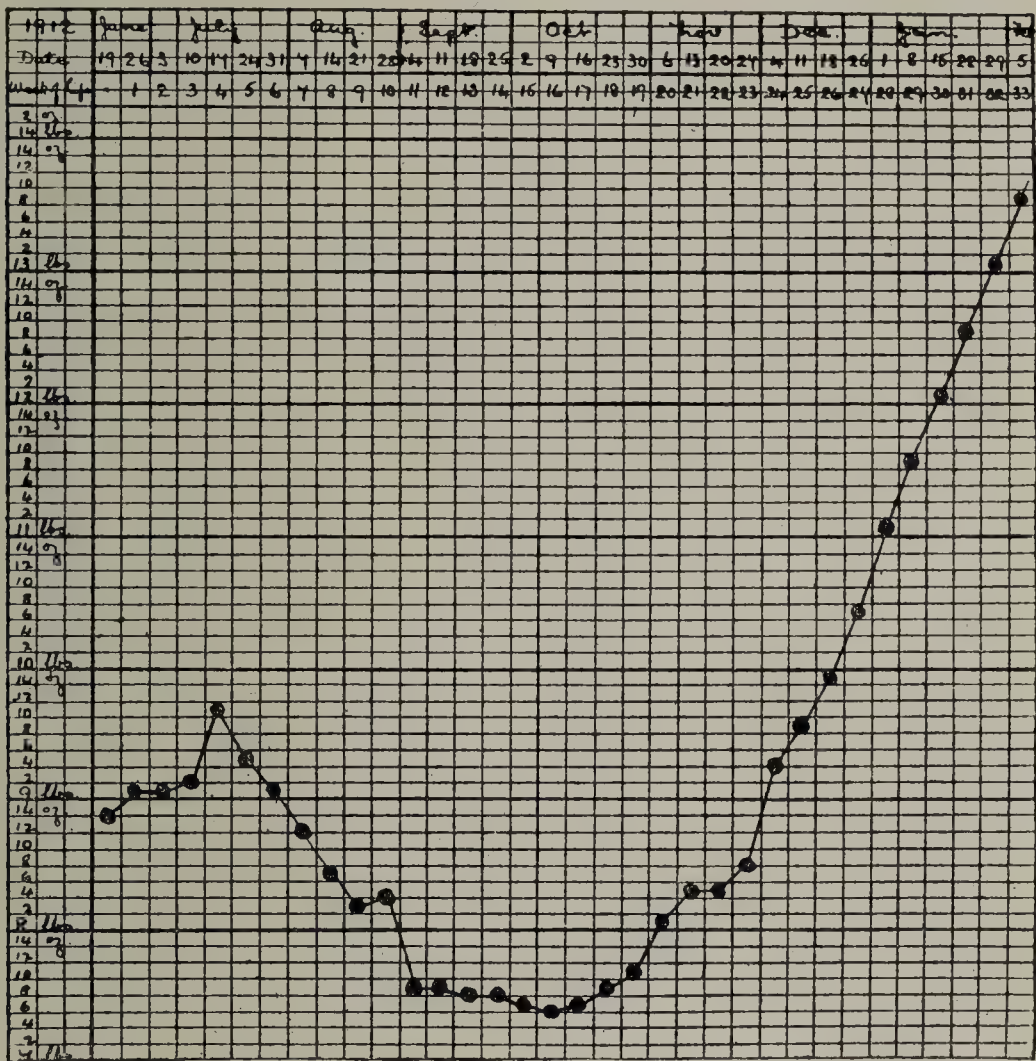


FIG. 112.—Weekly Weight Chart from medically treated case of Congenital Pyloric Hypertrophy.

Whether or not medical measures will be successful depends on the severity of the condition. Even when ultimately successful the recuperation in these circumstances is delayed, and the period till vomiting ceases and increase in weight sets in is an anxious one. The child is marantic and hence unduly susceptible to intercurrent infections, which may bring about a fatal issue at any time. Thus the stage of the illness at which one sees the case for the first time influences the decision whether medical or surgical measures should be adopted.

If the child is seen early in the course of the disease, *e.g.*,

during the first two weeks, operation is unhesitatingly the method of choice, and any medical measures adopted are simply for the purpose of rendering the patient more able to withstand the shock of the operation. But, if the condition is only recognised after an illness of many weeks' duration, medical treatment should be seriously considered, since we know that of its own accord the spastic obstructive state of the pyloric canal will in all probability soon disappear.

(a) *Medical Measures*.—In all cases, unless perhaps those seen in the first few days after the onset, medical measures should be adopted in the first instance. In the very early case the child is usually in a sufficiently good state for immediate operation. But with this exception, even although operation is already decided upon, it is wise to treat the child medically for twenty-four hours at least. This consists in lavage of the stomach, the administration of a suitable diet, and the treatment of any dehydration (anhydræmia—loss of fluid) which may be present.

Lavage of the stomach is one of the easiest of procedures. The child is wrapped in a blanket in order to control the movements of the limbs and is laid on his side on the nurse's lap. A No. 12 œsophageal catheter is the most convenient size and it is better if there be only one opening at the distal extremity. The catheter is lubricated with glycerine and pushed to the back of the throat when the child commences swallowing movements, so that it can be easily passed into the stomach. One knows by the escape of its contents when the stomach has been entered. There is, at least after the first occasion, very little retching during this operation. After the stomach contents have escaped a funnel with rubber-tube is attached to the catheter by means of a piece of glass-tubing which acts as a window. The funnel is then filled with luke-warm water containing one drachm of bicarbonate of soda to the pint, and by lowering and raising the funnel the stomach is washed out by siphon action and the process continued till the fluid from the stomach returns clear. The amount of water required before this happens varies much in different cases. In some instances one pint is sufficient, whereas in others several pints are necessary.

During this process of lavage of the stomach, by observing the amount of stomach contents which can be siphoned off in the first instance, the amount of fluid which the stomach can

hold, and the amount of fluid required to remove its contents completely, the degree of dilatation of the stomach and the delay in emptying (gastric motility) can be estimated.

The most suitable diet is of course breast-milk, and if this is available it is advisable that it be extracted and given in measured quantities by bottle rather than that the child should be permitted to drink direct from the breast. As a rule, however, the breast has been discarded before the condition has been recognised, and the child put on some milk mixture, on the assumption that the breast-milk was unsuitable. In these circumstances peptonised milk will be found most serviceable and given in the quantities and at the intervals normal for its age (see pp. 166 and 176). For directions regarding the preparation of peptonised milk, see p. 182.

All sorts of variations in feeding have been recommended in this disease—small quantities frequently and large quantities infrequently. In our experience, feeds of normal volume every 3 or 4 hours have given the best results. The thick feed recommended by Sauer¹ we have never found of any use and indeed, as a rule, distinctly harmful.

In cases of some duration the emaciation and dehydration may be extreme. Fluid subcutaneously, intraperitoneally, or intravenously, along with glucose, should be administered as described on p. 221. Blood-transfusion is recommended by some pædiatricians, but in our experience this procedure does not seem to have any special value.

Drugs of an antispasmodic nature, *e.g.*, atropine and morphia, we have found of little benefit.

(b) *Surgical Measures*.—If the case is seen early immediate operation is advisable. In all others, except those of very long duration and fast approaching the time of natural recovery, surgical intervention should be resorted to if, within two or three days of medical treatment, matters are either not improved or the child is not holding his own. Probably most physicians to-day would recommend operation in all cases except those of very long duration. But all are agreed that this may be delayed with advantage for twenty-four hours so as to give time by means of saline transfusions to improve the child's general condition. Unless the child is definitely anæmic blood transfusion is not followed by any better results than is saline transfusion.

¹ L. W. Sauer, *Arch. of Pediatrics*, 1924, xli., 145.

The operations which have been performed for pyloric stenosis are four in number, but the one practised by most surgeons to-day is Rammstedt's. This involves least manipulation and can be performed most quickly, which is a matter of prime importance. Rammstedt's operation can be completed from start to finish by any experienced surgeon in ten minutes.

Gastro-enterostomy was first performed with success by Lobker¹ in 1898, and shortly afterwards (1899) by Kehr²; forcible stretching of the pylorus (Loreta's operation) was introduced by Nicoll³ of Glasgow in 1900; pyloroplasty was first performed by Dent⁴ in 1902. It was in 1913 that Rammstedt⁵ of Munster first performed the operation which goes by his name; this consists in simple division of the circular muscular layer of the pylorus without incising the mucous membrane and without subsequent stitching. The only point in the operation which requires particular attention is to be sure that the muscular layer at the duodenal end is completely divided; otherwise complete relief from the obstruction will not result. While attempting this stage of the operation the bowel may be opened. If, however, this accident is recognised and dealt with, there is no superadded gravity to the operation and no special precautions during the after-treatment are called for. Recently Browne⁶ has remarked that attention must also be directed to the proximal or gastric end of the incision, since if this is not carried sufficiently into the wall of the stomach incomplete relief of the obstruction is apt to result. On account of both of these errors the operation has, on rare occasions, had to be repeated.

Whatever the operation performed the type of anæsthesia is a matter of importance. Gas and oxygen or local anæsthesia are generally admitted to give the best results.

Equal in importance with the anæsthetic is the after-treatment. Immediately after the operation it is advisable to give 1 minim of nepenthe by the mouth to induce sleep, and

¹ Lobker, *Verhandl. d. deutsch. Gesellsch. f. Chirurgie*, 29 Kongress, 1900, i., 148.

² H. Kehr, *ibid.*, p. 124; and W. Abel, *Münch. med. Wochenschr.*, 1899, xlviii., 1607.

³ J. H. Nicoll, *Brit. Med. Journ.*, 1900, ii., 571.

⁴ E. Cautley and C. T. Dent, *Med. Chir. Trans. Lond.*, 1903, lxxxvi., 474.

⁵ C. Rammstedt, *Zentralbl. f. Chirurgie*, Leipzig, 1913, xl., 3.

⁶ D. Browne, *Arch. Dis. Child.*, 1931, vi., 129.

fluid may be administered subcutaneously. Nutrient enemata— $\frac{3}{4}$ ii glucose in $\frac{3}{4}$ ss to $\frac{3}{4}$ i water—may be given, but unless these are of very moderate bulk ($\frac{3}{4}$ ss to $\frac{3}{4}$ i) and infrequently administered (every four to six hours) they are not retained. Food by the mouth can be commenced one hour after the completion of the operation. Breast-milk is the best, but if this is not available peptonised milk should be employed. Whatever the diet it should be given at first in small quantities and frequently. Since little food had been entering the bowel prior to the operation excessive amounts are apt to set up enteritis, which is one of the most grave complications. The following routine has been found satisfactory. Commence with 1 drm. and increase every alternate feed by 1 drm. up to $\frac{1}{2}$ oz. Then the period between the feeds is lengthened to one and a half hours and again every alternate feed is increased by 1 drm. to 1 oz. Then the period is further lengthened to two hours, and every alternate feed increased by 1 drm. to $1\frac{1}{2}$ oz. The interval between the feeds is now changed to two and a half hours, and alternate feeds increased by 2 drm. to 2 oz., which is given every three hours. During the following twenty-four hours the feeds are increased to 3 oz. every three hours, but one feed during the night is omitted, *i.e.*, seven feeds per twenty-four hours. In this way the child receives 4 oz. during the first twelve hours after the operation, 5 oz. during the succeeding twelve hours, 8 oz. during the two following periods of twelve hours, and then $8\frac{1}{2}$ oz. By the fourth day the child is receiving 21 oz. of milk in seven 3 oz. feeds.

The subcutaneous saline transfusions may be continued for the first few days, and of course water *ad libitum* may be given to drink. Normal saline is sometimes given instead of plain water, but it is questionable if it has any special advantage, while it has the definite disadvantage that it tends to cause œdema.

The results obtained in private practice are much better than those gained in hospital practice. Both John Thomson¹ and the present editor² have recorded their experiences demonstrating this fact. There would appear to be several reasons which account for this. In the first place, in private practice the patients are brought under the care of the physician

¹ J. Thomson, *Contributions to Medical and Biological Research*, New York, 1919, p. 1000.

² L. Findlay, *Brit. Journ. Child. Dis.*, 1923, xx., 1.

at an earlier stage of the illness, and consequently in a better nutritional state. In addition, the private patients receive much more individual attention.

Duodenal Atresia or Stenosis¹ is a not very rare condition and, as previously mentioned, may cause a picture simulating that of hypertrophic pyloric stenosis. The symptoms of obstruction, however, usually appear earlier (during the first few days), there is less marked gastric peristalsis, bile is present in the vomitus, and no pyloric tumour is palpable. These facts



FIG. 113.—Radiogram 21 hours after barium meal in case of Congenital Stenosis of Duodenum, which is seen greatly distended and filled partially with air and the opaque substance. (Girl aged 3 weeks.)

should raise suspicion regarding the true nature of the lesion, and an X-ray examination after a barium meal will reveal a characteristic picture (Fig. 113). There will be marked ballooning of duodenum and a passage or not of the meal into the small intestine, depending on whether the obstruction is complete or incomplete.

The obstruction is usually situated at the junction of the middle and lower thirds of the duodenum, and may be due to intrinsic or extrinsic causes.² Belonging to the former type of lesion are (a) atresia, (b) simple narrowing, and (c) a complete

¹ W. Sheldon, *Arch. Dis. Child.*, 1926, i., 279.

² T. T. Higgins and D. Paterson, *ibid.*, 1926, i., 285.

or incomplete diaphragm. The extrinsic causes are adhesions, bands, and aberrant vessels, with abnormal mobility of the viscus as a contributory factor¹ (Fig. 114).

It is impossible clinically to do more than differentiate between the complete and incomplete types of obstruction, but as recoveries are recorded in examples even where there was a complete hiatus in the continuity of the gut,² all cases should be submitted to operation. Within recent years quite a number of successful operations have been reported.



FIG. 114.—Stomach from case of Congenital Stenosis, with Dilatation of Duodenum.

Congenital Obliteration of the Small Intestine may occur in many situations, and may be single or multiple. Its commonest site is near the junction of the duodenum and jejunum. In this case, there is enormous distension of the portion of bowel above the obliteration, and well-marked visible peristalsis may occur during life (Fig. 115). The bowel below the obstruction is empty and abnormally small.

One of us (L. F.) has seen the first three children of a young married couple suffer from congenital obliteration of the small intestine.

An operation may be attempted, but the small diameter of the bowel makes it very difficult if not impossible. When nothing is done the child may live for about a fortnight.

Meckel's Diverticulum.—The persistent remnant of the omphalo-mesenteric duct is sometimes found as a blind tube from $\frac{1}{2}$ to 2 inches long coming off from the bowel about a foot

¹ C. P. Lapage, A. E. Somerford, and F. Howe, *Arch. Dis. Child.*, vi., 307.

² G. Bruton Sweet and C. Robertson, *ibid.*, 1927, ii., 186.

above the ileo-cæcal valve; or its umbilical end may be open, forming a fæcal fistula. When this is so, prolapse of the mucous membrane may occur, and this in infants gives rise to a form of umbilical polypus; it is easily cured by operation. In some cases the duct becomes acutely inflamed. In older children with this malformation, intestinal obstruction or strangulation sometimes occurs from constriction of the bowel by the diverticulum which has remained fixed to the abdominal wall.

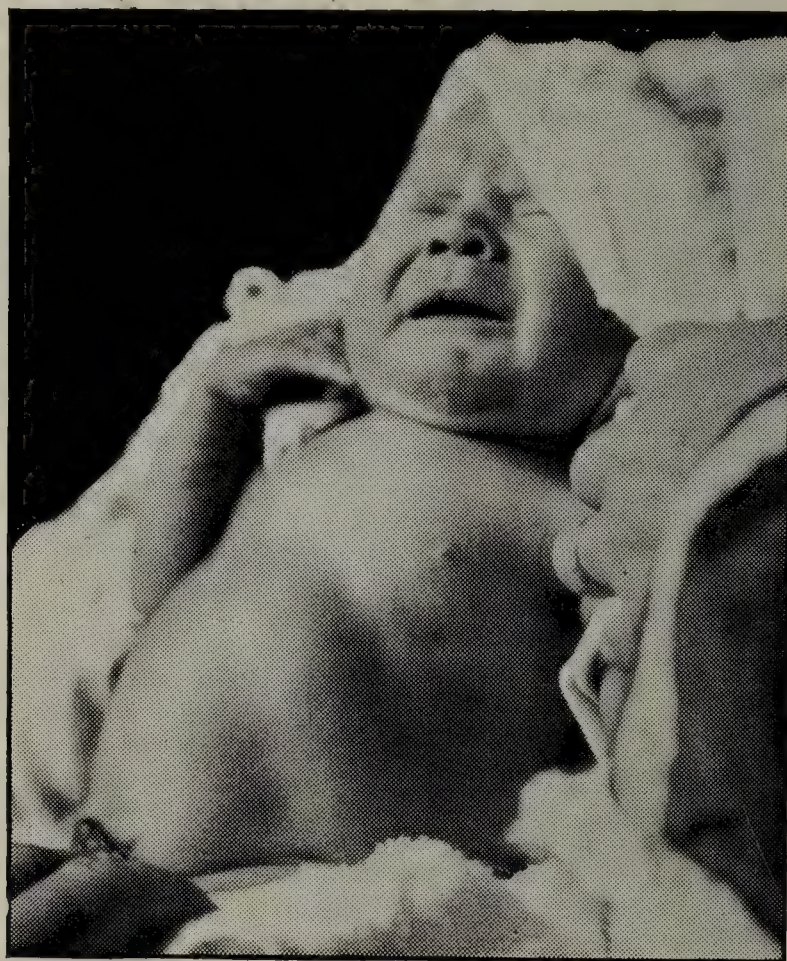


FIG. 115.—Child with Congenital Obliteration of Small Intestine.

In the slighter forms of **Imperforate Anus** in which only the external opening of the bowel is lacking, a simple incision and dilatation often give permanently satisfactory results. When the bowel ends at some distance from the anal orifice, however, the operation is much more difficult and its result often less satisfactory.

Slight Congenital Narrowing of the Rectum near its lower end is an occasional and important cause of obstinate constipation, and is apt to be overlooked unless a digital examination is made. In some of these cases the abdominal distension is such that they are mistaken for abdominal tuberculosis. The condition can be satisfactorily treated by a slight plastic operation.

Congenital Functional Derangements.**Congenital Hypertrophic Dilatation of the Colon***(Hirschsprung's Disease).*

In examining cases of great distension of the abdomen of long standing, it is well to bear Hirschsprung's disease in mind. In this condition there is enormous dilatation of the colon, with great thickening of its wall from hypertrophy of the muscular coat. Sometimes the whole lower bowel is affected,



FIG. 116.—Congenital Hypertrophic Dilatation of the Colon.

but often only a portion of the colon is enlarged. In the most typical cases there is no stricture or other obstruction to be found below the dilated part.

Symptoms.—The child, who is nearly always a boy, suffers usually though not always from *extreme* constipation, which dates from birth or, less commonly, from a few weeks after birth. The abdomen may not be very large at first, but it soon increases in size, and in time may become enormous, displacing the thoracic organs upwards, and ultimately, in some cases, causing œdema of the lower limbs. As the abdomen grows larger, the hypertrophied bowel is noticed from time to time to stand out on the surface (Fig. 116), and exaggerated peristaltic movements are seen passing along it.

At first, and sometimes for years, the child's general health is little affected. Afterwards, however, he usually becomes thin and feeble, and shows signs of dyspepsia and toxic absorption from the intestinal tract. In the later stages, which may be reached within the first year or two, but generally not until later childhood and sometimes not till adult life, there is diarrhœa with large slate-coloured, offensive, liquid stools, and often some rise of temperature. These terminal symptoms probably coincide with the appearance of extensive ulceration in the bowel—the result of the prolonged fæcal retention. Death takes place with symptoms of exhaustion and sepsis, and with dyspnœa from the pressure upwards of the distended bowel. In cases which survive to adult life there is a great risk of volvulus occurring.

The **pathology** of the condition is still obscure, though many theories have been formed to explain it. It seems probable that the primary defect lies in the nervous mechanism of the bowel. If such a defect existed which interfered with the co-ordination of the peristalsis, so that one part of the muscular apparatus constantly worked against another instead of in harmony with it, the muscular hypertrophy might be readily explained as the result of the consequent habitual overwork.¹ The "fibrillation" which occurs in the wall of the auricles may possibly be in some respects an analogous condition. In some cases there appears to be an obstruction at the recto-sigmoid junction and the dilatation ceases abruptly at that point.

Diagnosis.—The history of the onset of the symptoms and the absence of ascites, glandular masses, or adhesions, usually serve to distinguish this condition easily from abdominal tuberculosis. It may be distinguished from cœliac disease by the state of the abdomen, the lesser degree of cachexia, and the character of the motions.

Diagnosis can be made by X-rays with or without a barium enema or by the fact that a very large simple enema can be retained without discomfort.

Prognosis.—Improvement under treatment is common; but it is not probable that complete recovery ever occurs.

¹ J. Thomson, "On Defective Co-ordination in Utero as a probable Factor of certain Congenital Malformations," *Brit. Med. Journ.*, 6th Sept. 1902, 678.

Treatment.—Careful *dietetic* and *medicinal* treatment is of considerable value in giving relief during the earlier stages. The bowels should be relieved every two or three days. For this purpose combinations of strychnine, belladonna, and aloes may be used, or magnesia, Carlsbad salts, or petroleum. An occasional dose of calomel is sometimes beneficial, and massage may also be useful. Later, recourse must be had to enemata. Extreme flatulent distension of the colon is sometimes greatly relieved by the passage of a long tube into the bowel.

Several *surgical* operations have been practised with a varying degree of success. The most successful procedure seems to consist in *colostomy*, to relieve the immediate symptoms, followed later by *resection of the dilated colon*. As the dilatation and hypertrophy often reach right down to the rectum, it may not be possible to do this completely, and subsequent over-distension of the portion left may, in that case, give rise to trouble.

Wade¹ of Australia, inspired by the work of Hunter and Royle, performed lumbar sympathectomy and records good results. Rankin and Learmonth² of the Mayo Clinic and Barrington-Ward³ have also reported improvement in consequence of this operation. In our opinion, however, this operation is still in the experimental stage.

¹ R. B. Wade and N. D. Royle, *Med. Journ. Australia*, 1927, i., 137 ; R. B. Wade, *Lancet*, 1930, i., 136.

² F. W. Rankin and J. R. Learmonth, *Ann. Surg.*, 1930, xcii., 710.

³ L. E. Barrington-Ward, *Proc. Roy. Soc. Med.*, 1932, xxv. (Child. Sect., 73).

CHAPTER XV

DISEASES OF THE LIVER

THE liver is a very important organ in young children, and relatively to the rest of the body it is about twice as large as in adults. Notwithstanding its size and its physiological importance it is, in temperate climates at least, much less frequently affected by disease in childhood than in later life. When we do meet with instances of hepatic disease in children, however, they are often cases of special interest; although, unfortunately, the interest is apt to centre more in the diagnosis and prognosis than in the treatment.

When a young baby is *jaundiced*, the first question we ask ourselves is whether it is merely a case of icterus neonatorum (physiological jaundice) or if there is a pathological cause for the symptom. In the latter case, we may have to do with catarrhal jaundice, with some form of infective disease, with congenital syphilitic affection of the liver and its ducts, with congenital obliteration of the bile ducts, with congenital family cholæmia, or possibly with a hepatic neoplasm. Jaundice is also a prominent symptom in Buhl's disease (fatty degeneration of new-born children) and in Winckel's disease (epidemic hæmoglobinuria). These two rare affections of newly-born children have been described as occurring in continental maternity hospitals, but are rarely, if ever, seen in this country.

Far the commonest form of jaundice in older children is the ordinary catarrhal variety which is often associated with symptoms of gastric catarrh. If the jaundice is prolonged, severe, or recurrent, we should think of such diseases as hepatic cirrhosis, "subacute" liver atrophy, congenital family cholæmia, and icterus hæmorrhagica; possibly also of hydatids of the liver or of gall-stones. Jaundice is also occasionally met with as an incident in the course of various other diseases such as abdominal tuberculosis and advancing abdominal tumours.

Icterus Neonatorum (*Physiological Jaundice*).

In new-born children the skin is of a more or less deep red colour all over the body. This gradually fades, leaving in most cases a yellow tinge. When the yellow discoloration is marked, it is spoken of as icterus neonatorum, or physiological jaundice. It is generally noticed first on the second or third day after birth, and is never present when the child is born. After increasing in depth for a day or two, it gradually diminishes, and is usually gone within a week or ten days. In rare cases it may persist for several weeks.

The explanation of this phenomenon is still obscure. It is certain, however, that it is a form of hepatogenous jaundice, and neither, as was formerly held, hæmatogenous in origin, nor merely a local discoloration due to the red of the hyperæmic skin turning yellow as a bruise does in the process of fading. It seems probable that at birth, owing to the active destruction of red blood-corpuscles, a specially large amount of highly pigmented bile is secreted, but how this finds its way into the general circulation is still undetermined.

The discoloration differs from that in ordinary obstructive jaundice, both in its distribution and in the order of its appearance. It is first seen on the face and chest, later on the sclerotics, and last of all on the hands and feet. Compared with the skin, the sclerotics are slightly affected and they may remain normal in colour, while in ordinary jaundice they are among the parts earliest and most deeply discoloured. The urine also generally remains quite normal in appearance and the fæces are always so. In other respects the child is perfectly well.

The diagnosis of icterus neonatorum rarely presents any difficulty. The absence of serious symptoms, the slight degree of the jaundice, the normal urine, and the coloured motions suffice to distinguish at once even extreme instances of this condition from cases of infective or catarrhal jaundice, and from those which depend on syphilitic or other disease of the liver or on congenital obliteration of the bile-ducts.

Jaundice from Umbilical Infection.

Infective jaundice is often due to streptococci or other organisms spreading by the umbilical into the portal vein and thence to the liver. Streptococcal inflammations of the pleura, peritoneum, or meninges may also be present.

The **symptoms** are local and general.¹ The former consist in suppuration of the umbilical wound, with redness and swelling of the surrounding skin. The latter, which set in during the first two days of life, are irritability, sleeplessness, refusal of the breast, vomiting, and green diarrhœa. The jaundice increases, and there is more or less cyanosis and sometimes œdema. The temperature reaches 102° to 104° F. Hæmorrhages, which may be fatal, sometimes take place from the umbilicus or bowel. When the general infection is severe, the infant always dies within a few days in a comatose condition.

The **treatment** is mainly prophylactic, and consists in scrupulous aseptic precautions in the management of the umbilical cord.

Jaundice from Intestinal Infection.

This is a commoner condition than that which originates in the umbilical wound. It may occur in epidemics or sporadically, and has been said to depend on infection by *Bacillus coli* and *Bacillus lacticus*.² The bile-ducts are normal and the liver and spleen are little, if at all, enlarged. The intestine shows a slight degree of desquamative enteritis.

The **symptoms** set in rapidly without apparent cause, the mother being quite healthy. The child refuses the breast, vomits, and may have a slight convulsion. Jaundice, cyanosis, and diarrhœa soon follow. The motions are not decolorised. The cyanosis is a characteristic feature. It usually modifies the yellow tint to a sort of bronze, and may mask it altogether. The diarrhœa is of a peculiar character; it is not severe and may therefore be overlooked. At first, there may be only three or four pale green alkaline or neutral motions in the twenty-four hours; these are passed without pain, and the abdomen

¹ Porak et Durante, *Arch. de Méd. d. Enf.*, Paris, 1901, iv., 321.

² Lesage et Demelin, "De l'ictère de nouveau-né," *Rev. de Méd.*, 1898, xviii., 1.

remains soft and natural. In fatal cases this condition of the stools continues until death. When recovery sets in, however, the character of the motions changes; they become more frequent (seven to ten) and are bright green in colour and acid in reaction. The general infection gives rise to drowsiness, slight fever, and emaciation. The duration of the disease is from three to twelve days.

The **diagnosis** of this form of infective jaundice depends on the absence of an umbilical lesion, on the lower range of the temperature (not much over 100° F.), and on the absence of other infective lesions such as pleurisy, peritonitis, and meningitis.

The **prognosis** is grave, the mortality being about 30 per cent. Severe cyanosis, high fever, and profound drowsiness are all unfavourable signs. The passage of an increasing number of highly bilious and acid stools is of good omen.

The **treatment** consists in attention to the general hygiene, the administration of small doses of calomel, and especially in the use of subcutaneous injections of normal saline solution. Great care must be taken in the disinfection of the motions.

Jaundice from Congenital Syphilis.

The common form of intercellular syphilitic cirrhosis with, or more usually without, miliary gummata, does not cause jaundice. Occasionally, however, a severe degree of jaundice is met with in syphilitic cases, as the result of pericholangitis or from obstructive disease of the bile-ducts.¹ One of us (L. F.) met with a case due to pressure on the common bile-duct by a gumma situated in the head of the pancreas.² In such cases there are usually hæmorrhages in some situations and death occurs with profound cachexia, in spite of anti-syphilitic treatment. The only features which distinguish the condition from congenital obliteration of the bile ducts (which has nothing to do with syphilis) are the presence of syphilitic symptoms in the parents, other indications of the disease in the child, and the severe cachexia.

¹ Beck, *Prag. Med. Wochenschr.*, 1884, ix., 257, 266, 284; H. D. Rolleston *Brit. Med. Journ.*, 1907, ii., 947.

² L. Findlay, *Syphilis in Childhood*, 1919, p. 70.

Congenital Obliteration of the Bile-Ducts.¹

Under the heading "congenital obliteration of the bile-ducts," a series of cases of congenital jaundice are described in which there is obstruction, or an actual disappearance of the larger bile-ducts, often with signs of past inflammation of these parts and with biliary cirrhosis of the liver.

Etiology.—The disease has occasionally been reported as occurring in several children in a family, and, in some instances, multiple malformations of other organs have been found along with it. There are generally no grounds at all for attributing the lesion to congenital syphilis, although a few cases of obstruction of the ducts due to this disease have been placed on record (Beck,² Rolleston³).

Pathology.—The degree to which the bile-ducts and gall-bladder are affected varies very greatly. In a few cases the ducts are only slightly obstructed or thickened; but usually their lumen is entirely obliterated or portions of them have entirely disappeared. The obliteration may be situated at any part of the ducts, or it may affect the gall-bladder only. The portion of the duct above the obliteration is sometimes greatly distended. In a few instances gall-stones composed of inspissated bile have been found in the ducts. The liver is always enlarged and in a state of biliary cirrhosis if the child has lived for any length of time. The pancreas may be affected with fibrosis, and the spleen is generally much enlarged. The inflammation in and around the ducts very rarely spreads to the peritoneum.

Whatever the cause of the condition it is of the nature of a chronic inflammation of the ducts with the development of fibrous tissue leading to their obliteration. It has been suggested that it is predisposed to by congenital narrowing. The consequent stagnation of the bile is responsible for the accompanying

¹ Beneke, *Die Entstellung der kongenitalen Atresie der grossen Gallengänge*, Universität Programm, Marburg, 1907; J. B. Holmes, *Amer. Journ. Dis. Child.*, June 1916, xi., No. 6, 405; L. S. Milne, *Quart. Journ. Med.*, July 1912, v., No. 20, 409; J. Thomson, *Edin. Med. Journ.*, 1891-92, xxxvii., 523, 604, 724, and 1112; and *Allbutt and Rolleston's Syst. of Med.*, 1908, iv., pt. 1, 103.

² *Prager Med. Wochenschr.*, 1884, ix., 257, 266, and 284.

³ *Brit. Med. Journ.*, 1907, ii., 947.

hepatic cirrhosis. Experimental ligation of the ducts leads to intercellular cirrhosis.¹

Clinical Features.—The parents are generally healthy, although, in some instances, they have had several other children affected with congenital jaundice apparently of the same nature. Boys are more frequently affected than girls (41 : 26).

The child is usually well nourished at birth and appears normal until the whiteness of the stools and the persistent jaundice are noticed. Jaundice, however, may be present from birth, but usually appears on the second or third, and occasionally not until the tenth or fourteenth day, or sometimes even later. It is slight at first, but steadily deepens, and the skin soon assumes a dark greenish yellow hue which persists till death, though it often varies a little in intensity from day to day. The motions are generally colourless from the first, but several cases have been reported in which meconium of the usual colour has been passed before the stools became white. The characteristic pale stools contain an excessive amount of fat (65·5 per cent.), due chiefly to soaps and fatty acids, as the splitting of the fat is unimpaired.² The Van den Bergh test gives a direct positive result. The urine is generally deeply bile-stained.

A characteristic complication in these cases is the occurrence of spontaneous hæmorrhages from various situations. Sometimes the bleeding is from the umbilicus (fifth to ninth day), and this form is usually fatal. Patients who survive the first fortnight may have bleeding into the subcutaneous tissues or from the nose, stomach, or bowel. When no complications occur, the disease seems to cause comparatively little interference with the general health and a considerable proportion of the children live for many months (three to ten); but no case in which the diagnosis was confirmed post-mortem has survived the first year. Towards the end, emaciation, debility, and sometimes convulsions occur.

The **diagnosis** may present some difficulty at first; but the colourless motions, the deeply bile-stained urine, and the increase of the jaundice during the second week, soon show

F. Fischler, *Ergeb. d. inn. Med. und Kinderh.*, 1909, iii., 240.

² H. Hutchison and G. B. Fleming, *Glasgow Med. Journ.*, 1920, xciv., 65.

that there is something more serious the matter than icterus neonatorum, and the absence of severe cachexia also differentiates the condition from infective and syphilitic disease, in which case the stools contain bile.

Treatment.—While medical treatment is quite useless, it is possible that relief might be obtained in a few instances by a surgical operation, if it were done early, before the liver has had time to become seriously diseased. From a consideration of the post-mortem conditions found in the published cases, Holmes concludes that 16 per cent. of them might have been operated on with, at least, some chance of success by making an anastomosis between the duodenum and the gall-bladder or a part of the bile-duct above the obliteration. Considering that the prognosis is otherwise hopeless, an operation of this kind may be regarded as justifiable, but the chances of its being successful are certainly very small indeed.

Congenital Family Cholæmia

(Congenital Acholuric Jaundice).

In examining obscure cases of jaundice in children, it is well to bear in mind the rare disease known as “congenital family cholæmia,” as its symptoms in many cases begin in infancy. The condition is almost certainly to be regarded as a form of hæmolytic jaundice arising from some congenital defect in the blood-forming organs—the disturbance of the liver function being merely secondary.¹ As it is apt, however, to be mistaken for forms of jaundice due to liver disease, it may conveniently be considered here. Its pathology and its relation to other infantile affections of the hæmopoietic organs are still very obscure. The clinical manifestations also vary in detail to a considerable extent according to different observers; and it seems not improbable that further study may reveal the existence of several distinct types of the disease.

Clinical Features.—The disease is in many cases hereditary and, when this is so, the sexes are equally affected and transmission occurs either through males or females, but never through members of the family who have no symptoms of the disease.

¹ F. J. Poynton, *Lancet*, 15th Jan. 1910, 153; A. W. Mackintosh, A. W. Falconer, and A. G. Anderson, *Edin. Med. Journ.*, Feb. 1911, 101.

The patients are curiously free from subjective symptoms, even when the degree of jaundice they present is considerable ; and the disease does not apparently lead to debility or tend to shorten life. The chief physical signs are great enlargement of the spleen, certain changes in the blood, and a degree of persistent jaundice.

The splenic enlargement is present from the beginning and the organ reaches to or even below the level of the umbilicus. In a few cases severe pain is complained of in the splenic region, which is probably due to dragging from the weight of the organ. The splenomegaly is due entirely to engorgement of the sinuses, this with blood pigment being the only change observed on histological examination.¹

The blood serum always contains urobilin, but, as a rule, no bilirubin. The red corpuscles are diminished in number and show a lowered resistance to hæmolysing agents such as saline solution ; there is often also a degree of poikilocytosis and microcytosis instead of the megalocytosis which is usual in obstructive jaundice ; nucleated red cells are common. The colour index is normal. The leucocytes are normal in number and in their relative proportions, but various abnormal forms are often found. Occasionally slight epistaxis or hæmorrhage from the gums or elsewhere is met with, but dangerous purpuric symptoms never occur.

Soon after birth, in most cases, a mild degree of jaundice begins to appear gradually, and this persists through life. It increases during the periodic exacerbations, but is never extremely severe. At these periods the liver is also enlarged, but at other times it is normal in size.

The urine is in most cases altogether free from bile pigment, but urobilin is generally to be found in it. In other respects it is quite normal. The fæces are always well coloured.

The exacerbations which occur from time to time with varying frequency are very characteristic of the disease. Sometimes there is no apparent cause for their onset, but they are occasionally due to excess in eating or drinking, to bodily or mental strain, or to exposure to cold or heat. In these attacks there is a slight rise in temperature, an increase in the jaundice, usually some enlargement of the liver, distinct increase in the

¹ W. C. MacCarty, *Proc. Staff Meetings Mayo Clinic*, 1932, vii., 187.

anæmia and in the size of the spleen, often also a heavy deposit of urates in the urine, and a deeper coloration of the fæces.

It has been shown that *splenectomy*, while not modifying the fragility of the red-blood corpuscles, brings about a cessation of their destruction and the consequent periodic attacks of anæmia and "jaundice."

Catarrhal Jaundice.

Catarrhal jaundice is a fairly common disease in older children. Occasionally it occurs as an epidemic in a family or school, without any apparent cause; and it is met with frequently in some influenza epidemics. Its clinical features are the same as in later life. In early infancy it is comparatively rare, but it may occur even during the first few months of life. At this age it is to be diagnosed by the absence of the special symptoms of the other forms of jaundice which occur in young infants, by the gastric symptoms which accompany it, and by the patient's recovery after a week or two. The Van den Bergh reaction is biphasic.

The treatment of catarrhal jaundice consists in confinement to bed, restriction of the fats and milk-curd in the food, and the administration of rhubarb, bismuth, and soda. The bowels, if costive, should be kept moving by a mild saline laxative and occasional doses of calomel.

Spirochætal Jaundice.

This disease has only appeared in Great Britain in recent years, and as yet it has been met with chiefly in adults, and especially in miners—the infection having been usually spread by rats.

A small outbreak, involving five children and two adolescents, which was observed in Edinburgh during 1924, has been reported by Prof. D. Murray Lyon.¹ From the clinical features of the cases it appears that, in young children especially, the disease may assume a mild form, and that it is very apt to spread from one child to another. It seems not improbable that some of the obscure outbreaks of jaundice observed in the past may have been of this nature.

The main facts of this small epidemic were as follows: A little girl of five came from Singapore to visit a family in

¹ *Lancet*, 7th March 1925, i., 504.

Edinburgh. During the voyage home she had been ailing a good deal, and when she arrived she was still out of sorts and slightly jaundiced, and her temperature reached 100° F. on several occasions. Some time after she left Edinburgh, spirochætes were found in her urine.

The family visited consisted of five children. Four of these, aged 7, 13, 17, and 19 years, took ill at intervals thereafter, and developed jaundice of various degrees of severity. The parents and servants were not affected. Soon after, a second family—friends of the first—took ill similarly, though less severely, their ages being 3, 6, and 10 years.

The *symptoms*, generally speaking, were as follows: On the first day, the child felt merely out of sorts. By the next morning, there was a furred tongue, headache, and vomiting, with or without diarrhœa, and a temperature of about 102° F. Pains in the limbs and body generally followed, with sometimes a degree of liver tenderness. Jaundice began about the fourth or fifth day. Small petechiæ appeared on the arms and abdomen in some of the cases; and, in two, there were ecchymoses on the palate. In one there was blood-stained vomit, and in three epistaxis. All the cases recovered. The only two who were seriously ill were the adolescents.

In most instances the spirochætes were found in the urine. The three most severe cases were treated with antispirochætal serum, and seemed to be much benefited by it.

Cirrhosis of the Liver.

Multilobular cirrhosis from prolonged ingestion of alcoholic liquors or other unknown irritants is occasionally observed in the child, but it is rare.¹ The symptoms are said to be similar to those in the adult, but the course of the disease is usually of shorter duration.

A *monolobular or biliary form* of the disease,² usually called "Hanot's Cirrhosis," which presents interesting peculiarities, is sometimes seen in older children. It is of unknown causation, and may occur in several members of a family.

The main clinical features are chronic jaundice lasting for

¹ E. Jones, *Brit. Journ. Child. Dis.*, 1907, iv., 43.

² Hanot, *La cirrhose hypertrophique avec ictère chronique*, Paris, 1892; James Finlayson, *Glasg. Hosp. Rep.*, 1899, 39.

years, with a uniform smooth enlargement of the liver and a very large spleen. Ascites is only found in the later stages. Repeated attacks of abdominal pain and tenderness with pyrexia and increased jaundice occur from time to time. Clubbing of the fingers often develops, and, towards the end, there is a hæmorrhagic tendency. The disease may last for years without causing any cachexia; but the patients are apt to be undergrown and anæmic. No treatment is known.

A *familial juvenile cirrhosis* of the liver with cirrhosis of the pancreas and spleen, and a type of anæmia simulating splenic anæmia (Banti's disease), is occasionally met with.¹

The *intercellular cirrhosis* caused by congenital syphilis is referred to elsewhere (p. 363).

Acute and "Subacute" Atrophy of the Liver.

Acute Atrophy of the Liver.—It is rare for acute liver atrophy of the ordinary classical type to occur in childhood. We have only seen two or three instances of this. Such cases may, however, occur at any age from early infancy onwards, and they run the same rapidly fatal course as in adults, generally ending in death within a week, or at most two, from the first appearance of the jaundice. The early symptoms resemble those of ordinary catarrhal jaundice and give rise to no anxiety. After a few days, however, the child becomes rapidly worse. The jaundice deepens and vomiting occurs, or, if previously present, assumes a more severe type, and blood may appear in the vomited matter and in the stools. Cerebral symptoms—delirium, drowsiness, and convulsions—soon set in and death follows in a state of coma. Subcutaneous hæmorrhages and epistaxis are common. The liver dullness rapidly diminishes. The urine is scanty and deeply bile-stained, and it may contain albumin; on evaporation, it shows a copious deposit of leucin and tyrosin crystals.

While this is the course taken in the most acute cases, a large majority of those which begin somewhat in this way assume a less severe type—the so-called "subacute atrophy"—and temporary recovery takes place (*v. infra*).

Subacute Atrophy of the Liver.—The term "subacute liver atrophy" has been applied to a type of case which

¹ F. G. Gunn, *Arch. of Path. and Laby. Med.*, 1926, i., 527.

would be more accurately described as "recurrent partial acute atrophy of the liver." The morbid process which occurs in it is not really subacute in the ordinary sense. It consists in a recurrent acute destruction of tissue which differs from what takes place in the acute liver atrophy merely in the fact that it affects severely only certain scattered areas of the liver tissue, and so much escapes destruction that the organ is able to carry on its functions, and a partial recovery results. It is possible that, when the proportion of the liver tissue destroyed is small, permanent recovery may sometimes occur. In most cases, however, there is a speedy, and in time a fatal, recurrence of the disease.

This type of liver atrophy is especially characteristic of childhood, probably owing to the more vigorous resistance which the child's tissues offer to the disease, and to their greater power of regeneration. During the course of fifteen years one of us (J. T.) met with nine cases of this kind, five of which were examined post-mortem. It seems probable that the condition is commoner than was formerly thought. Certainly many fatal cases of jaundice have been diagnosed as hepatic cirrhosis from other causes which were really instances of this disease. No definite line of distinction can be drawn between the milder cases of acute and the severer instances of subacute atrophy, as the difference depends solely on the extent to which the liver tissue is involved.

It is not necessary to discuss here the very obscure subject of the pathology of acute liver atrophy or its microscopical anatomy. It will be sufficient to say that the characteristic necrosis of the cells occurs in a partial and patchy manner throughout the organ and varies greatly in amount; and that the place of the destroyed cells is rapidly taken by fibrous tissue, while the liver endeavours to make good its loss of tissue by very active hyperplasia of those areas which have escaped destruction.

The naked-eye appearance of the liver is striking. Owing to the shrinking of the affected areas, and the bulging of those which have escaped and are undergoing hypertrophy, the surface is exceedingly irregular and nodular. The nodules vary in size and are of a bright green or a yellow colour, the intervening shrunken areas being reddish brown or pinkish grey.

Microscopic sections of the nodules show extremely hyper-

plastic liver cells, which often contain two or more nuclei, and many smaller cells which are evidently newly formed. The reddish shrunken tissue between the nodules contains no proper liver cells, but is made up mainly of young cellular newly-formed fibrous tissue and blood vessels, and has a number of "bile-duct-like" structures ramifying through it.

Symptoms.—The clinical manifestations of subacute liver atrophy vary considerably in different cases. The main symptoms in the cases observed may be summarised as follows:—

The *onset* of the jaundice and the severe nervous symptoms were usually preceded by headache, vomiting, and loss of appetite for a varying number of days; and these often continued after it appeared. In two of the cases there was diarrhoea also at the beginning of the illness. The *duration* of the attack varied greatly. It lasted generally for at least two to eight weeks, but in two of the cases the jaundice had persisted for five months at the time the child was first seen. The *number* of attacks also varied. In two cases (both of which were subjected to post-mortem examination) the patient died in the first attack. In the remainder it was the second, third, or fourth attack which proved fatal.

Jaundice is generally, but not invariably present. In two of the cases there had been two previous attacks in which there was jaundice, but this symptom was entirely absent in the third. In both of them, however, all the other chief symptoms were present, including severe pain in the right hypochondrium, fever, drowsiness passing on to unconsciousness, and ascites; and in one which was examined post-mortem the condition of the liver was typical.

Fever was present in four of the cases (102° to 104°), and absent in the other five. *Restlessness*, *delirium*, and increasing *unconsciousness* were prominent symptoms in five of the children, and two of them had blowing stertorous breathing not unlike that in uræmia. A *tendency to hæmorrhage* showed itself in three cases. In one there was epistaxis, in another bleeding from the gums, and in the third subcutaneous ecchymoses. *Severe abdominal pain*, generally in the right hypochondrium, was complained of in at least five of the nine cases. A considerable degree of *ascites* was present in three of them.

The *liver* was enlarged (from 1 to 4 f.b.) in seven of the

cases; in the other two it was normal or slightly diminished in size. The *spleen* was more or less greatly enlarged in five, and normal in size in the others.

The *stools*, in all, were either very pale yellow or colourless. The *urine* was bile-stained. In two instances it contained albumin. In none of the cases was leucin or tyrosin discovered on evaporation.

Treatment.—The only treatment which can be recommended in liver atrophy is the repeated subcutaneous injection of saline solution.¹ This is sometimes followed by improvement in the cerebral symptoms. It probably acts by aiding the elimination of toxic substances.

Gall-Stones.

Gall-stones are very rare in childhood.² In most of the published cases they occurred in young infants and consisted of comparatively soft masses of inspissated bile. In some instances their formation seems to be merely an incident in the course of congenital obliteration of the bile-ducts.³

Malignant Tumours of the Liver.

Malignant growths in the liver in young children are usually sarcomatous in nature; they are not at all common. The most striking symptoms are the extremely rapid growth of the liver, which in some cases can be noticed to enlarge from day to day attacks of severe pain in the hepatic region, and irregular rises of temperature, along with no apparent cachexia and no obvious interference with the general nutrition. Usually there is neither jaundice nor ascites, and the spleen is not enlarged. The disease is rapidly fatal.

Cysts of the Liver.

Simple Cysts.—A large single cyst of the liver is occasionally met with. If it projects downwards from the lower aspect of the organ it may be mistaken for a right hydronephrosis. There

¹ H. Morley Fletcher, *Garrod, Batten, and Thursfield's Dis. of Child.*, London, 1913, 210.

² Mercat, *De la colique hépatique chez l'enfant*, Thèse de Paris, 1884; Gourdin Servenière, *De la lithiase biliaire dans l'enfance*, Thèse de Paris, 1889; Still, *Trans. Path. Soc. Lond.*, 1899, l., 151; Stoeltzner, *Med. Klinik.*, 1909, No. 1, 1.

³ J. Thomson, *Edin. Hosp. Rep.*, 1898, v., 1.

is usually no jaundice. The treatment consists in incision and drainage; and the prognosis is good. A cystic condition of the liver is occasionally associated with a similar condition in the kidney (congenital cystic kidney).

Hydatid cysts of the liver are almost unknown in Scotland; but cases are sometimes seen in older children from Orkney or Shetland. The symptoms, diagnosis, and treatment are the same as in later life.

Fatty Liver.

Fatty infiltration of the liver is very common in early life, and is responsible for a considerable proportion of the enlarged livers found in little children. On clinical examination the fatty liver often feels so hard that it is thought to be either cirrhotic or amyloid.

Fatty degeneration of the liver is also common. It is seen, in its most extreme degree, in delayed chloroform poisoning and other forms of acid intoxication.

CHAPTER XVI

THE SKIN

THE condition of the skin as to colour, moisture, and texture, as well as temperature, should always be carefully noted, and the presence of any œdema, desquamation, eruption, or other abnormality investigated. A thoroughly healthy skin usually means a child that is not very ill.

Colour.

Jaundice.—The various causes and varieties of jaundice have been considered in the last chapter.

Cyanosis.—General cyanosis may be a sign of disease of the heart or lungs, either congenital or acquired. When it occurs in an acute illness, it is usually of importance as indicating heart failure or serious pulmonary disease. Obscure recurrent attacks of blueness in an infant may, in rare instances, be due to abnormalities outside the heart, such as diaphragmatic hernia (p. 849). Cyanosis may also be present in diseases characterised by extreme prostration, such as septicæmia and malignant cases of infectious disease. Lividity of the extremities is frequent in all kinds of wasting diseases, and is especially marked in Raynaud's disease (p. 596).

Pallor.—The peculiar pallid brownish-yellow tinge of the skin which is characteristic of splenic anæmia is often seen in children, especially if they have been much in the open air; but it is entirely absent in many cases in which the spleen is much enlarged. The sallow, earthy tint of some scorbutic infants is a help in diagnosing the condition; and the same may be said in the case of syphilitic babies.

Pigmentation.—Addison's disease is very rare in children; but a brown discoloration, especially marked about the axillæ, groins, abdomen, and perineum, is common in chorea and other diseases when arsenic has been given in large doses for some

time. Leonard Guthrie pointed out that pigmentation of the skin is "a most important diagnostic sign of interstitial nephritis in both old and young, although not existing invariably."¹

Patchy pigmentation of the skin may occur as an isolated phenomenon, and sometimes it is a symptom of a general disease, as in the case of the *café au lait* spots found in von Recklinghausen's disease (generalised neuro-fibromatosis).

An interesting form of congenital pigmentation of the skin is the so-called "**Mongolian Blue Spot**" in the sacral region or elsewhere on the back, which is found in 90 per cent. of Japanese babies at birth. These spots are said to be frequently present in all the dark races. C. Herman² found them in 25 per cent. of negro infants, and also in 1 in 400 of white babies. The spots gradually fade as the child grows, and are invisible in a few years.

Nævoid Patches.—A reddish patch on the back of the child's neck, about the level where the hair begins, is not very rarely complained of by mothers. It is due to a slight nævoid enlargement of superficial vessels, and is often so faint that it is entirely overlooked until the patient, if a girl, begins to "put up her hair." It is not amenable to treatment.

Reticular Mottling of the Skin (*Livedo Annularis* or *Cutis Marmorata*).—The peculiar mottled appearance which exposure to cold brings out in many normal skins is commoner and more distinct in childhood than in adult life, and ill-nourished children with languid circulation are especially apt to show it. The condition is, up to a certain point, to be regarded as physiological; it owes its pattern to the distribution of the deep venous plexus. In some morbid states, such as scrofula, syphilis, rheumatism, myxœdema, and especially mongolism, it is often present to an unusual degree. In those who are predisposed to it there is sometimes a tendency for other skin eruptions, such as those of measles, syphilis, and tuberculosis, to take on a marbled pattern of this kind to some extent.³

A brownish "measling" of the skin with the same marbled appearance is sometimes seen, generally as the result of intermittent exposure to strong, dry heat. In it the mottling is persistent and pigmented, and does not disappear on pressure,

¹ *Lancet*, 27th Feb. 1897, i., 587.

² *Journ. of Cutan. Dis.*, May 1907, 201.

³ H. G. Adamson, *Brit. Journ. of Dermat.*, 1916, xxviii., 281.

owing to exudation having taken place from the blood vessels. The presence of this eruption may occasionally be helpful in practice. For example, in children with various cachectic conditions, we find it, not very rarely, on the shins and on the inner side of the knees and thighs (Fig. 117). This usually shows that the child has been in the habit of crouching closely over a fire to relieve a distressing feeling of chilliness. When a similar condition is met with in other parts of the body,



FIG. 117.—Reticular Mottling of the Skin.

it may indicate the position of a very severe pain which has been made bearable by the repeated use of hot bottles or some other form of dry heat.

Lineæ Albicantes (*Striæ distensæ*).—These are occasionally found in older children in various situations, and usually follow distension of the skin from rapid accumulation of fat or from the occurrence of œdema. When they first appear they are pink in colour, but they become white in the course of time. Henoch¹ draws attention to their occurrence across the front of the knees in typhoid. In this situation they are believed to be

¹ Henoch, *Lect. on Children's Dis.*, 4th edit., New Syd. Soc. Transl., 1889, ii., 348.

due to the rapid growth of the bones and to the child having been lying long with his knees flexed.

Moisture and Dryness.

Profuse perspiration in infancy is nearly always due to rickets, although it may also occur in severe tuberculosis, in erythrœdema, and in other toxic and suppurative conditions. In older children it is most frequently a symptom of intestinal indigestion, and calls for a thorough regulation of the diet and general tonic measures, such as more fresh air and cold douching. Small doses of oxide of zinc (1 to 2 gr.) are also sometimes useful. It occurs rarely in childhood as a symptom of phthisis pulmonalis.

Texture.

The skin in healthy children is remarkably soft, elastic, and pliable. These characters are, of course, interfered with by such congenital conditions as ichthyosis; but if the skin of a sick child has been formerly normal, and has only recently lost its softness and resiliency, this should always be noted, as it may be an omen of serious significance.

A dry, harsh skin is met with in babies with digestive disorders when the vital powers are being dangerously strained. In older children its presence may sometimes draw our attention, in cases of tuberculosis, diabetes, and chronic renal disease, to the serious nature of an illness when this has hitherto been overlooked. A dry, harsh condition of the skin is also met with in older cases of mongolism and of untreated cretinism.

Ichthyosis.—Slight cases of ichthyosis in which there are dryness and thickening of the epidermic layer of the skin are of common occurrence. The condition is always congenital, and often also hereditary in origin, but generally it does not show itself for some weeks or months after birth. In rare instances, it is noticed from the first.

The treatment of all the forms of ichthyosis consists in removing the scales by alkaline and bran baths, and using habitually some form of fatty ointment. Kaposi recommended a 5-per cent. ointment of β -naphthol along with naphthol soap.

If such measures are diligently employed, they greatly improve the state of the skin and the patient's comfort for the time; but whenever they are discontinued the condition becomes as bad as ever.

Tylosis (*Hyperkeratosis*) **Palmæ et Plantæ**.—This is a rare family disease which shows itself in early childhood and affects both sexes equally.¹ It consists in a great thickening of the horny layer of the skin on the palms and soles, which is sometimes associated with hyperidrosis of these parts. At certain times of the year this thickened epidermis peels off, leaving the underlying skin red and very tender. Many of the children affected are mentally deficient.

The treatment consists in removing the thickened epidermis by means of starch poultices and soaking with carbonate of soda, and in the local application of salicylic acid either as a 3-per cent. plaster or in the form of an ointment ($\frac{1}{2}$ dr. to 1 oz.) combined with ichthyol (1 dr.). If this line of treatment is persistently carried out, the disease can be kept in check to a large extent; but the condition relapses rapidly whenever it is neglected even for a short time.

Desquamation.

Any peeling of the skin is worthy of attention. If its distribution is bilateral or general, it suggests, of course, a suspicion of scarlet fever; but it not infrequently occurs in other diseases, such as rubella and typhoid, and after some toxic and drug rashes. In hospital patients, it may perhaps sometimes be favoured by the application of soap and water to an unwonted extent. In children suffering from prolonged feverish ailments, such as tuberculous affections of the mesenteric and bronchial glands, we often find a branny desquamation of the epidermis (*pityriasis tabescentium*).

Desquamation limited to the chest is often due to rube-facient applications and poultices. In infants of a few months, scaling of the palms and soles is an important symptom in congenital syphilis. Erysipelas, also, is generally followed by profuse desquamation.

¹ Lewis Thatcher, *Edin. Med. Journ.*, 1912, N.S., viii., 342.

Œdema.

Although most forms of œdema of the skin are only part of a general dropsy, the subject of œdema may conveniently be considered in this chapter. It may either be localised or more or less general in distribution.

Localised Œdema may occur (*a*) from *lymphatic obstruction*, after an attack of erysipelas or in connection with various other forms of inflammation of the skin or deeper parts. It may also be due to (*b*) temporary or permanent *obstruction in large veins*. For example, slight œdema of the eyelids is characteristic of severe whooping-cough; in infantile scurvy, dropsy of the eyelids and of the limbs is a commoner occurrence; and local swelling of this kind in the hands and feet is seen in severe cases of infantile tetany, and sometimes in purpura.

Another variety is (*c*) *angioneurotic œdema*, which is a form of giant urticaria. This sort of localised dropsy is supposed to depend on an anaphylactic process through the abnormal fluids acting directly on the muscle fibres or blood vessels. It is sometimes an alarming condition. Acute swelling of the cheek, hand, scrotum, or some other part of the body appears suddenly and passes off again rapidly, often in the course of an hour or two. Although there is generally no danger connected with it, there is always a possibility of the swelling extending to the fauces and giving rise to fatal œdema glottidis. This, however, very rarely happens. When the dropsy affects one of the lower limbs in an infant it may simulate infantile scurvy. The treatment consists in stopping the elements in the diet which are disagreeing with the child, if they can be identified.

Generalised Œdema may be set up in various ways. It may be caused (*a*) by *general venous obstruction* from heart failure (cardiac œdema). This shows itself mainly in the most dependent parts. It occurs much less commonly in children than in adults, and is only seen in them in the later stages of acquired heart disease. Œdema also occurs (*b*) in *anæmia* and *cachexia* from hydræmic plethora. This form of it also affects dependent parts chiefly. (*c*) Œdema is, of course, a common symptom in *kidney disease*, especially in acute primary and in chronic parenchymatous nephritis; and whenever it is

present, the urine must be carefully examined. Occasionally, in cases of acute nephritis, severe dropsy may set in before albuminuria has begun. In renal dropsy the peculiar distribution of the swelling depends more on the local laxity of the tissues than on gravity.

One of the commonest and most interesting forms of œdema in young infants is that seen in (*d*) *defective excretion of salts* from the body. As already mentioned (pp. 134 and 136), an excess of salt (and also of sugar) in a young infant's diet is apt, even in health and still more during sickness, to lead to a gain in weight from increased retention of fluid in the tissues. The retention of salt is not due to any defect on the part of the kidneys, which continue to excrete phosphates, urea, and other substances in the usual way. This kind of dropsy may be only temporary, but it usually takes several days before diuresis is sufficiently established to get rid of the extra salt. In most of the cases there is no noticeable superficial œdema, but, in new-born and premature infants, the condition may go on to obvious dropsy of the skin; and the younger the child the longer does the swelling take to disappear. It sometimes occurs when beef-tea or bouillon is given to a weakly baby who is being fed on a weak milk mixture or on breast-milk. In infantile atrophy œdema is specially prone to occur. It is also not uncommon in any babies of a few weeks old who are being overfed.

The management of this form of œdema consists in the thorough dietetic treatment of the nutritional disturbance (Chapter IX.), and not in the mere withholding of salt, as is indicated in renal dropsy. When the food is properly regulated the œdema disappears; and, naturally, the less salt and sugar there is in it the more rapidly does recovery take place. When the treatment is begun, a rapid loss of weight is to be expected. Any attempt to lessen the œdema in such cases by the administration of diaphoretics, diuretics, or digitalis is harmful as well as useless.

When œdema occurs in the course of chronic diarrhœa in young children, the hypodermic administration of adrenalin solution in doses of 5 minims twice daily seems often to accelerate the disappearance of the swelling.¹

¹ W. E. Hume, *Brit. Med. Journ.*, 1911, ii., 478.

Œdema Neonatorum.—When a baby is born œdematous, or becomes so immediately after birth, this may be due to congenital nephritis. The majority of slight cases of congenital dropsy, however, depend on other causes; such a condition usually indicates a dangerous degree of weakness.

Sclerema Neonatorum.

True sclerema neonatorum is a rare disease, met with almost exclusively in continental maternity and foundling hospitals. The children may seem normal when born, but they are usually very feeble and often premature. Within a day or two of birth they begin to waste, the temperature falls to below 90° F.—sometimes as low as 84° F. or even 78° F.—in the rectum, and the skin becomes thickened and hard. Beginning in the lower limbs, the induration spreads over the trunk, arms, and face, and the whole body may become stiff with it. The surface is of a pale, dirty yellow colour, and cold like that of a dead body; the extremities are cyanosed; the thickened parts do not pit on pressure. Death almost always takes place in three or four days, or within three or four weeks at latest.

Pseudo-Sclerema.

The term “pseudo-sclerema” may be applied to a type of case which is not very uncommon in this country, and which is often mistaken for true sclerema. It resembles the latter in causing large indurated areas of skin and subcutaneous tissue with a similar but usually less extensive distribution.

The affected areas of skin are of a purplish-red colour, apparently painless, very much thickened and quite hard, so that they do not pit at all on pressure. The temperature varies from a little below to a little above normal; but there is no tendency to the extreme subnormal temperature which is characteristic of true sclerema, and no collapse. The children usually recover under treatment, but it is often several months before all induration has gone. Those cases which we have seen recover were treated by inunction with either mercurial or ichthyol ointment along with careful attention to the general health. It seems probable that this is not a condition of the

same nature as real sclerema. The distribution and character of the lesion, and its usual course, make it more probable that it is an infantile form of *diffuse symmetrical scleroderma* which is met with in rare instances in older children.¹

Subcutaneous Emphysema.²

In young children subcutaneous emphysema sometimes occurs in the neck and neighbouring parts, as a consequence of violent coughing (Figs. 118 and 119). It may or may not be



FIG. 118.—Subcutaneous Emphysema, following severe coughing.

accompanied by pneumothorax. The air is driven upwards from the mediastinum by the cough, and finds its way into the subcutaneous tissues of the neck, face, scalp, and sometimes further. It usually occurs in cases of measles, whooping-cough, or bronchitis; and it is generally recovered from, unless the original condition is serious. When recovery takes place, the air is absorbed in from three to seven days. The main treatment consists in soothing the cough.

Another cause of subcutaneous emphysema is the puncture, with a large exploring needle, of a lung which is adherent to the chest wall. Unless local pressure is applied at the time, the

¹ H. Radcliffe Crocker, *Diseases of the Skin*, 2nd edit., 1893, 371.

² L. Guillemot, *Grancher and Comby's Traité des Maladies de l'Enfance*, 2nd edit., iii., 498; Huber and Hirsch, *Arch. of Ped.*, March 1910, xxvii., 191.

air may spread considerably. This sometimes gives rise to a rise of temperature (about 101° F.) for two or three days, but it need cause no anxiety.

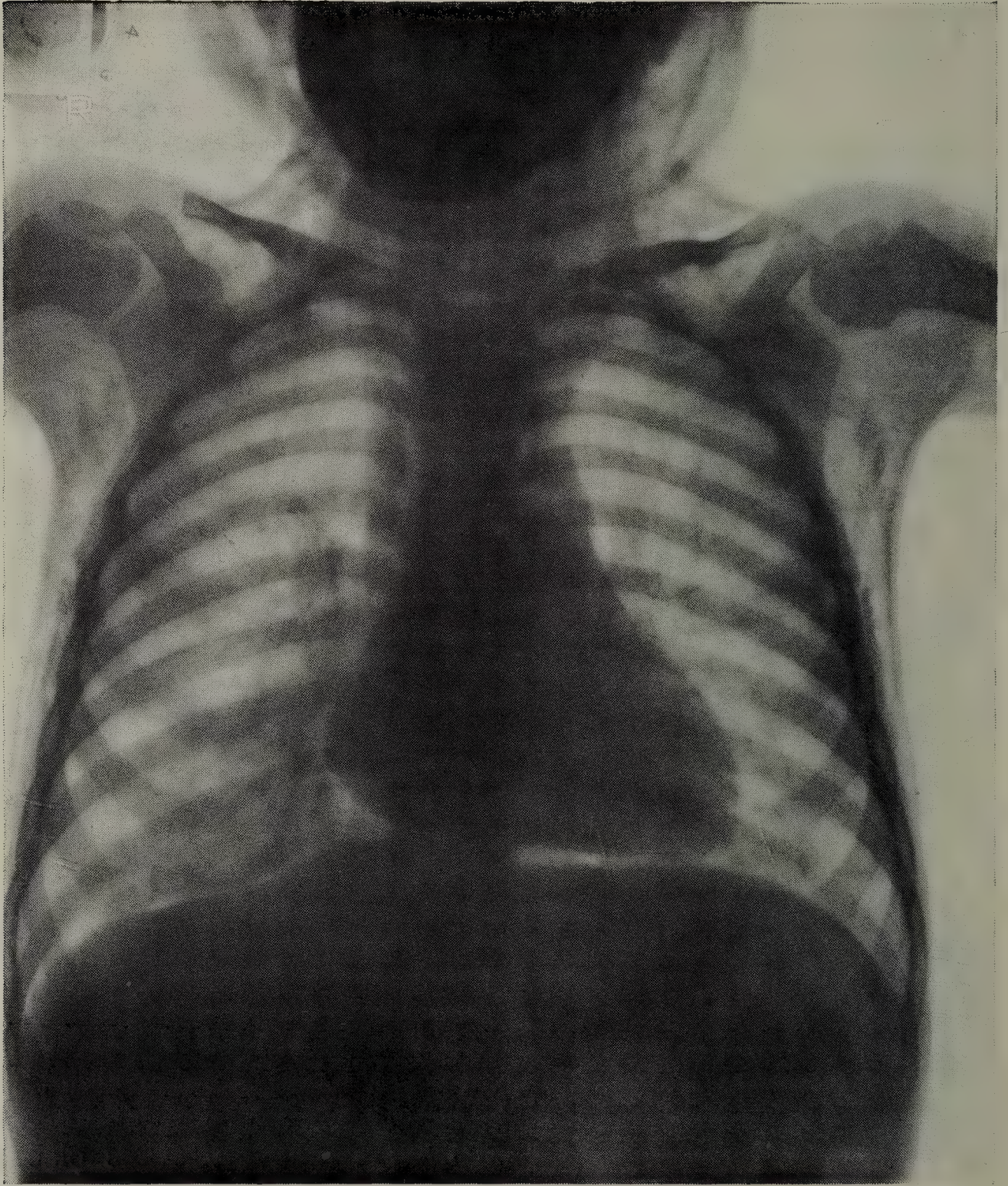


FIG. 119.—Radiogram of example of Mediastinal and Subcutaneous Emphysema following severe coughing in broncho-pneumonia. Note delimitation of cardiac shadow from diaphragmatic shadow. (Boy $4\frac{3}{4}$ years.)

Eruptions.

Skin eruptions are to be investigated as to their distribution, the presence of itchiness, the anatomical character of the lesions, and their apparent cause.

Distribution.—The precise position and extent of the rash are the first points to be ascertained, as they may throw much light on its cause. In early infancy, for example, the palms and soles are favourite sites for the lesions of congenital syphilis and scabies. Eczema is met with mostly on the face, and never on the napkin area; while the latter region is the favourite site for erythema from the irritation of ammoniacal urine, streptococcal eruptions, seborrhœic dermatitis, and specific eruptions. The napkins irritate the convex areas of the skin which are in contact with them, and spare the flexures. The syphilitic rashes usually affect the convex surfaces and the flexures equally. Other peculiarities in the distribution of specific rashes will be referred to later (Chapter XXXVI., p. 917).

Itchiness.—A common and distressing symptom is itchiness, which occurs especially in urticaria, prurigo, eczema, erythroedema, chilblains, scabies, and pediculosis, sometimes also in the early stages of measles and scarlet fever, and in varicella. It is never present in syphilitic rashes.

Character of the Lesions.—In infancy, according to Radcliffe Crocker, skin eruptions “are more likely to take a pustular form, and, from the ease with which the alimentary canal is deranged, there is greater tendency to eczema or urticaria.” Rutherford Morison has drawn attention to the fact that *cellulitis* is almost unknown in early childhood. It is important to remember this, because cases of acute osteomyelitis are sometimes mistaken at first for cellulitis and the necessary operation deferred with disastrous results.

Causation.—In considering the causation of any extensive eruption in a young baby, the possibility of congenital syphilis, scabies, and drug eruptions should never be forgotten. The effect in exciting and aggravating skin disease which is exerted by scratching, excessive perspiration, washing with strongly alkaline soaps, imperfect drying of the skin after bathing, and exposure to strong winds, must also be remembered. The influence of teething on eczematous eruptions has been already discussed (p. 50). Vaccination, although often unjustly blamed, certainly does occasionally act as an exciting cause of skin eruptions, and generally tends to aggravate them if present; in rare cases, however, it may have a beneficial effect upon a very chronic eczema. The influence of general toxic con-

ditions of all kinds in causing skin eruptions is always to be borne in mind.

Infantile Eczema.

This is a not uncommon condition occurring in children between six and eighteen months of age, and is variously called "*infantile eczema*," "*eczema seborrhoicum*," and "*seborrhæic dermatitis*." Since it presents varying appearances from different degrees of severity and the true cause is still unknown, the old and familiar designation "*infantile eczema*" seems at present as suitable as any.

Causation.—There are a possible number of etiological factors which may be at work in causing eczema. There are *local exciting* causes such as scratching, dirt, excessive perspiration, the use of strong soaps, and exposure to strong, dry winds. These rapidly bring out the eruption on some skins, although in most children they have no such effect; and even in the same patients do not always do so. Then there are *general predisposing* causes which are often equally important. We have already seen how strong an influence teething has in starting and prolonging eczema in many cases, and how vaccination and other acute ailments may also occasionally predispose to it; but what is of more importance is the strong temporary predisposition sometimes set up by errors of diet and digestive disturbances.¹ Thus, there is a form of acute exudative eczema in thriving breast-fed babies who are gaining weight rapidly, which is caused simply by excessive feeding, and only needs restriction of the breast-milk for its cure. Similarly, in bottle-fed babies and older children, examination of the stools, and diminution of those elements of the food which are found not to be properly digested, may cause rapid improvement and disappearance of the eruption. The elements which are mostly to blame in these cases are animal fats and starches. When digestive disturbances are followed by eczema, this may be due to incomplete digestion, or a catarrhal condition of the mucous membrane causing direct absorption of proteins into the blood, thereby rendering them capable of acting as antigens. The fact that local and general causes which

¹ Harvey P. Towle and Fritz B. Talbot, *Amer. Journ. Dis. Child.*, Oct. 1912, x., 219; Rowland G. Freeman, *Arch. of Ped.*, 1914, xxxi., No. 70, 735.

evidently produce eczema in some children do not do so in others, shows, however, that some further predisposing influence exists. This may consist in a congenitally defective formation of the skin, but probably more frequently depends on an inherited constitutional abnormality.

There can be no doubt that infantile eczema is sometimes, like urticaria and erythema, to be regarded as a clinical manifestation of modified anaphylaxis (allergy); and, as Schloss,¹ Hoobler² and others have shown, it is certainly sometimes set up by the absorption of some unaltered protein, such as egg-albumin, from the alimentary canal. A large number of children who have eczema in early infancy develop asthmatic symptoms later; about one-third of the children with asthma whom one of us (J. T.) has treated were said to have previously suffered from eczema. It is also interesting to note that infantile eczema often runs in families, and that it is also not uncommon to find a history of gout, asthma, hay-fever, and idiosyncrasies to common forms of food, among the patient's near relatives.

Barber³ describes two main types of eczema, apart from that produced by external irritants. The *first* of these corresponds to the true seborrhœic eczema of adults. "It affects the scalp, the post-auricular regions, the face round the nose and mouth, the neck, and the flexures. Its onset is often sudden, and oozing takes place very rapidly, so that secondary infection occurs early, resulting in impetiginisation. . . . This form of eczema is apparently associated with excessive carbohydrate feeding." The *second* type is probably always due to susceptibility to one or more foreign proteins—usually, of course, those of food substances, but in a few cases to bacterial infection. These children are generally emotional and neurotic, even in early infancy, and are apt to develop asthma later. Their skin is often more or less ichthyotic. "The eczema takes the form of diffuse scaly patches, which become thickened; they are situated on the forehead, the cheeks, the extensor surfaces of the limbs, the trunk, and sometimes the flexures. Oozing does not readily occur, although it is often provoked by scratching, nor is secondary impetiginisation nearly so common as in the

¹ *Amer. Journ. Dis. Child.*, 1912, iii., 341.

² *Ibid.*, 1916, viii., 129.

³ *Brit. Med. Journ.*, 8th Oct. 1921, ii., 557.

first type. One very characteristic feature is that the eczematous patches may become quite pale and almost invisible in the course of a few hours, only to flare up when a paroxysm of itching or a fit of crying occurs. This is not the case in the first type, in which, too, the itching is not so intense."

Prognosis.—The chances of recovery are generally good. Most cases can be much improved, and many cured, by a few weeks of careful external and internal treatment; though the disease is apt to recur. It is important to remember that this tendency of the eruption to return passes off, in most cases, when the process of dentition is complete.

In all cases in which a large part of the body surface is covered for a long time by an eczematous eruption, we must bear in mind the risk there is of septic contamination, and also the danger which arises if the kidneys become affected while the skin is in such a state. Cases are occasionally reported in which children with extensive eczema die suddenly with high temperature and convulsions. The explanation of these cases is still obscure.

Treatment. (a) *General.*—In treating a case of infantile eczema, the first thing to do is to revise the diet, and to see to it that the child is not being overfed and is having plenty of fluid. We may then ascertain, by inspecting the stools, that he is not having more fat or carbohydrate than he is able to digest. If constipation exists, it should be relieved by dieting or the regular use of laxatives; and diarrhœa, if present, should be *gradually* checked. In our experience, however, dietetic measures have had little effect.

Generally, internal medication is not indicated; but, if the child is anæmic or rickety, iron or cod-liver oil may be very useful. If he shows signs of oxaluria, the addition of citrate of soda to the milk, or giving small doses of salol and sodium bicarbonate, is often very beneficial. Arsenic is rarely of any use, and generally aggravates the inflammation in acute cases. In these, small doses of antimonial wine (1 to 2 minims) are sometimes recommended. In a few very chronic eczemas the administration of thyroid sometimes has a good effect.

Bromides are of little value in relieving the itching of eczema. The most useful drug for this purpose is antipyrine (1 to 2 gr. in babies); and it is sometimes well to secure an occasional good night by giving a full dose of chloral. The

essential point in the treatment of the itching, however, when the free application of ointment does not relieve it, is to secure the child's hands by tying them to the bed, or by applying arm-splints in such a way that scratching is made impossible.

(b) *Local*.—The first point in the local treatment of the condition is to protect the diseased skin from irritating influences. The child must be carefully kept out of strong winds. High bracing places will generally be found bad for him. Washing with strongly alkaline soap should be forbidden. The affected parts must, of course, be kept clean, but the bath should be of short duration and carefully given, and oatmeal or bran and water or normal saline solution is better than plain water. When some soap is found necessary, an over-fatty variety should be chosen. After drying, a zinc and starch paste is useful. Ointment should be spread preferably on old linen, and bandaged on; when the eczema is on the face it should be applied on a mask (Fig. 120).



FIG. 120.—Mask for Facial Eczema.

The best local application to select depends on the state of the eruption. In cases with much crust-formation it is always well to begin with starch and boracic poultices, which soften the scabs, so that they can be easily removed, and lessen the inflammation. Later, a calamine lotion (Appendix E, Form. 51) is often useful. Lassar's paste, and white precipitate ointment (5 gr. to 1 oz.) with or without the addition of zinc oxide (1 drm. to 1 oz.) are favourite applications. The best preparation in our hands has been the ointment of the oleate of bismuth (B.P.). Very acute cases are often benefited by the addition of ichthyol to the ointment (20 gr. to 1 oz.). Eczema behind the ears generally recovers rapidly with an ointment of bismuth and zinc (āā 1 drm. to 1 oz.), or of tannic acid (1 drm. to 1 oz.). In seborrhœic eczema a small amount of sulphur (5 gr. to 1 oz.) should be added to the ointment. In chronic cases, when the skin is very dry, great relief may often be given by soaking with Carron or cod-liver oil.

When the itching is severe—provided the eruption is not moist—the application of tar in some form or other is generally indicated. It may be used as a lotion (liq. picis carbonis 1 drm. to 10 oz. of water), or as an ointment (ung. picis 1 drm., ung. zinci to 1 oz.). If any preparation of tar is being applied to a large surface of skin, a watch must be kept on the urine, as carboluria or albuminuria may be set up.

Erythema of the “Napkin Area.”

The region covered by the napkins is often the site of acute erythema set up by ammonia given off by the urine. The cause of this is discussed elsewhere (p. 426). The eruption is confined to the convex surfaces of the skin which are in contact with the diapers, and it spares the flexures. The calves and heels are often affected.

The *treatment* consists in the careful washing and frequent changing of the napkins. The affected skin should be repeatedly washed with warm water or saline solution, and vaseline or zinc ointment applied. In the more severe cases an ichthyol ointment is useful. The child's digestion should be attended to.

Erythema Nodosum.

Symptoms.—Erythema nodosum is a common condition in childhood. Its well-known red, rounded, slightly raised and tender areas are generally met with on the shins only, although sometimes they occur on the extensor surface of the arms also, and to a much less extent on other parts. The eruption usually lasts for two or three weeks, but relapses sometimes occur. It is important to bear in mind that it may be preceded and accompanied by a remittent temperature closely resembling that of early typhoid and by considerable disturbance of the general health. Occasionally “rheumatic” joint pains are complained of; hence the eruption was formerly regarded as a manifestation of rheumatism.

The etiology of the disease is still obscure, but it seems certain that it may result from various infective agents, although Lendon¹ has suggested that it is a specific infection which he would call “nodal fever.” It has frequently been ascribed to the

¹ *Nodal Fever*, London, 1905.

rheumatic infection, but it is only very seldom related to this disease. According to French opinion, tuberculosis is the most common etiological factor; Pons¹ reported the presence of giant-cell formation and Landouzy² that of the tubercle bacillus in the centre of the nodule. The benign nature of the malady is, however, strong evidence against it being an active tuberculous lesion. Boganovitch³ has found it a frequent sequela of influenza. In America⁴ the view is held that it is of streptococcal origin.

It is a self-limited disease which, after persisting for some days, invariably subsides and gets completely well. It is very doubtful if treatment is of any avail. If there is much pain and fever, confinement to bed, a milk diet, and saline laxatives may be prescribed. Aspirin may be given for the relief of pain and as a febrifuge. It is probably in this way that salicylate of soda also acts.

Erythema Pernio (*Chilblains*).

Chilblains are common in cold, damp weather, in delicate children, and also in many who do not seem delicate apart from their sluggish circulation. Bluish red patches appear on the toes, heels, and margins of the soles, on the fingers and ears, and sometimes, in babies, on the cheeks. The patches are tender and cause severe itching, and burning, especially when the parts are warm. In neglected cases, and when there is pressure from badly-fitting boots, the skin is apt to break and extensive ulceration may occur.

Preventive *treatment* is important as, once the chilblains have formed, remedies are only partially successful. Children who are subject to the complaint should be encouraged to take much active exercise in the open air, and to accustom themselves to sleep with open windows. The habit of warming the feet and hands at the fire should be avoided as it is particularly harmful. Modified cold douching is useful. Strong, *roomy* boots with warm stockings are essential. The general health must be attended to, and there are several medicines which are

¹ J. Pons, *Thèse de Lyon*, 1905-6, No. 57.

² L. Landouzy, *Presse Méd. Paris*, 1913, xxi., 941.

³ V. Boganovitch, *Arch. Dis. Child.*, 1930, v., 56.

⁴ J. H. Hess and S. L. Berman, *Med. Clin. North America*, Phil., 1928, xii., 49.

sometimes useful. Calcium lactate (5 to 10 gr. every four hours for several days) is generally recommended, but it is doubtful if it exerts any definite influence on the condition. Cod-liver oil is also prescribed, and small doses of liquor arsenicalis are said sometimes to have a distinctly prophylactic effect. Small doses of thyroid (1 gr. twice daily) should be tried in obstinate cases. In girls of low intellectual capacity, about the time of puberty, this treatment is specially indicated, as in them a temporary degree of hypothyroidism is not uncommon.

When the chilblains are unbroken, tincture of iodine may be painted on once daily, or a weak capsicum ointment (capsici $\frac{1}{2}$ drm., ol. amygd. 2 drm., lanolini 6 drm.) rubbed in. When the skin is broken, boric or zinc ointment, alone or with a drachm to the ounce of ichthyol or of resin ointment added, may be used. The application of a Bier's bandage round the wrist or ankle for a few minutes once a day, followed by rapid removal, is also sometimes useful. Broken and suppurating chilblains may sometimes be greatly benefited by the use of an autogenous vaccine.

Enema Rash.

The administration of a rectal injection occasionally causes a rash. It consists of slightly raised bright red patches of erythema which are small and rounded at first but afterwards run into blotches. It is commonest in children between six and twelve years old.

The distribution of the eruption tends to be symmetrical and is characteristic.¹ It is found on the body, on the extensor surface of the elbows and knees, on the buttocks, and on the face—especially the cheeks and chin, but never on the palms and soles. Occasionally it is preceded by a diffuse scarlatini-form rash which affects the face as well as the body. It is accompanied by little or no itching. The interval between the administration of the enema and the onset of the eruption varies from two and a half to forty-eight hours (usually twelve to twenty-four), and the rash remains visible from twelve to seventy-two hours (usually twenty-four to forty-eight). There is no constitutional disturbance, no rise of temperature, and no enlargement of glands. There is generally no desquamation.

¹ G. F. Still, *Clin. Soc. Trans. Lond.*, 1899, xxxii., 11.

The rash is most apt to appear after a first enema, and usually, though not always, soap has been used. A similar eruption may follow a saline purge. No treatment is necessary.

Erythrœdema (*Pink Disease*).

In 1903 Selter of Cassel (Germany) described under the title of "tropho-dermatoneurose" or "vegetative neurosis" eight examples of this condition. This communication escaped attention and general interest in the disease dates from 1914, when H. Swift of Adelaide recorded fourteen cases and suggested for it the name of "erythrœdema." During recent years a number of papers have appeared from various parts of the world, describing its symptoms and dealing with the pathology and treatment.¹

Clinical Features.—The disease may occur at any age between three months and four years; it has not been recognised in older children. Most of the published cases have been between nine months and two years old.

The earliest symptoms are obstinate refusal to take food of any kind, followed by a loss of weight, distressing restlessness, wakefulness, and extreme irritability. Photophobia is a characteristic feature, so that the child tends to bury his face in the pillow or in the breast of his nurse. These symptoms may, or may not, have followed a slight attack of coryza or bronchial catarrh. There is no tendency to spontaneous vomiting. The stools may be loose, but they are more often constipated, and they usually seem fairly well digested. If the child is induced to take a sufficient amount of food, no great emaciation occurs.

A week or two after the loss of appetite, profuse perspiration

¹ P. Selter, *Arch. f. Kinderh.*, 1926-27, lxxx., 244; H. Swift, *Austral. Med. Cong.* (Children's Sect.), 1914; Byfield, "A Polyneuritic Syndrome resembling Pellagra-Acrodynia," *Amer. Journ. Dis. Child.*, Nov. 1920, xx., 347; A. Jeffreys Wood, "Erythrœdema," *Med. Journ. of Austral.*, 1921, i., 145; F. Parkes Weber, "Case of Erythrœdema (The Pink Disease)," *Brit. Journ. Child. Dis.*, Jan. to Mar., 1922, xix., 17; H. Thursfield and D. H. Paterson, *ibid.*, 27; E. Feer (Zurich), "Eine eigenartige Neurose des vegetativen Systems beim Kleinkinde," *Ergebn. d. inneren Med. u. Kinderheilk.*, 1923, xxiv., 100; D. Paterson and Godwin Greenfield, "Erythrœdema Polyneuritis," *Quart. Journ. Med.*, Oct. 1923, No. 65, 6; G. M. Findlay and R. O. Stern, *Arch. Dis. Child.*, 1929, iv., 1; W. G. Wyllie and R. O. Stern, *Arch. Dis. Child.*, 1931, vi., 137.

sets in and continues both day and night apart from exertion or overheating. This is soon followed by a very irritable sweat rash on the trunk and elsewhere. A few days, or in some cases many weeks, later, a bluish-red erythematous rash appears on the hands and feet. It is accompanied by distinct swelling, but by no real œdema, and followed by profuse desquamation. The rash begins on the fingers and spreads upwards, but very rarely passes beyond the wrists; at its margins it has a finely papular character. The feet are affected in like manner. Erythematous patches of a similar nature may appear on the cheeks, chin, and nose. The extent and degree of the eruption vary greatly in different cases. There is no rise of temperature in uncomplicated cases, and the red areas are quite cold to the touch. The itching of the body and limbs is extremely distressing, and the scratching often leads to excoriations, and sometimes to deep ulceration or sloughing of the skin. In some cases the nails fall off from time to time; and although there seems to be no itchiness of the scalp, many of the children have a habit of pulling out their hair in large quantities, and others scratch their ears until they bleed.

Chronic catarrh of the naso-pharynx is often present; and stomatitis is also common and may be preceded or accompanied by falling out of the teeth.

The muscles suffer early and become very soft and atonic, so that the child soon loses whatever power he has had of sitting up or standing and his head tends to fall forward on his chest; there is, however, no actual paralysis. The knee-jerks are often absent. The sensibility to pin-pricks is usually lessened or lost, but such of the children as can speak sometimes complain of tenderness when pressure is made on the limbs.

Although apathetic and dull at times, he is generally in a state of constant restlessness, extremely ill-tempered, and always pathetically wretched—usually there are no other mental symptoms.

Frequency of micturition has sometimes been observed; and slight albuminuria is not uncommon. Mild attacks of *Bacillus coli* pyelitis have often been noted; and starvation from the refusal of food not infrequently leads to acetonæmia.

The blood usually presents a leucocytosis of from 15,000 to 40,000; the proportion of polymorpho-nuclears being normal, or

nearly so. The heart and lungs are not usually affected; but the pulse is rapid and the blood pressure high.

Progress and Prognosis.—The disease generally lasts from three to nine months, and in a large majority of the cases gradual recovery takes place. Once the patient has recovered there seems to be no tendency for the disease to recur. When the child has not been induced to take enough food, however, death may result from debility and heart failure; more frequently intercurrent attacks of broncho-pneumonia or septic complications set in, and they may prove fatal.

Diagnosis.—Before the characteristic rash appears it is always difficult to recognise the condition with certainty; but, when the clinical features have had time to develop, they are so characteristic that the diagnosis can scarcely be overlooked.

Etiology and Pathology.—The time of year has apparently no influence. It is not a deficiency disease, for it may set in in thriving breast babies, and in those of the well-to-do, as well as among the poor. It is doubtful how far the disease is predisposed to by influenza. It has no connection with syphilis or pellagra; and as yet there seems to be no sufficient grounds for attributing it to the action of a special micro-organism. Although many typical cases have been reported from Great Britain, America, and the Continent, there seems to be little doubt that the disease is far more prevalent in the Australian cities than elsewhere; but no reason for this has been suggested.

Paterson and Greenfield describe the characteristic lesion in the nervous system as “a peripheral neuritis mainly affecting the sensory nerves, and showing a secondary vasomotor disturbance of the extremities.” In one of their cases they found post-mortem evidence of extensive polyneuritis and other microscopic changes in the nervous system. Whether these lesions are primary or secondary seems as yet not quite certain.

Treatment.—Wyllie and Stern record very good results by the addition of raw liver to the diet (2 to 3 oz. daily). This treatment was suggested by the experimental work of Findlay and Stern. These workers produced in rats a syndrome simulating that of Pink Disease by special dietetic measures, and found that it could be hindered or rapidly cured by the addition of liver to the diet.

The children must have plenty of fresh air, but they must be kept very warm, and all unnecessary fatigue avoided. The

most important indication (Byfield) is to control the anorexia by spoon-feeding, or, if necessary, by the use of the stomach-tube. Any food that is swallowed seems to be fairly well digested. Tonics such as *nux vomica*, arsenic, and iron have also been recommended.

Feer found that large doses of calcium salts had sometimes a soothing effect in the early stages; and in one case he observed very marked benefit from the use of atropin (2 mg. pro d.). The sweating lessened, the hands became warmer and of a better colour, and the child was less irritable and slept better. In two of our cases the injection of an autogenous vaccine, prepared from the stools, was followed by improvement, but this may have been merely a coincidence.

The chief local treatment of the skin consists in the prevention of scratching by the use of elbow-splints or otherwise. If the skin is not broken, rubbing with methylated spirit or painting with tincture of iodine may be tried; and if abrasions have occurred, the application of a zinc and tar ointment.

J. S. Fowler has found painting with a 1-per cent. solution of chromic acid the best thing to use for the stomatitis.

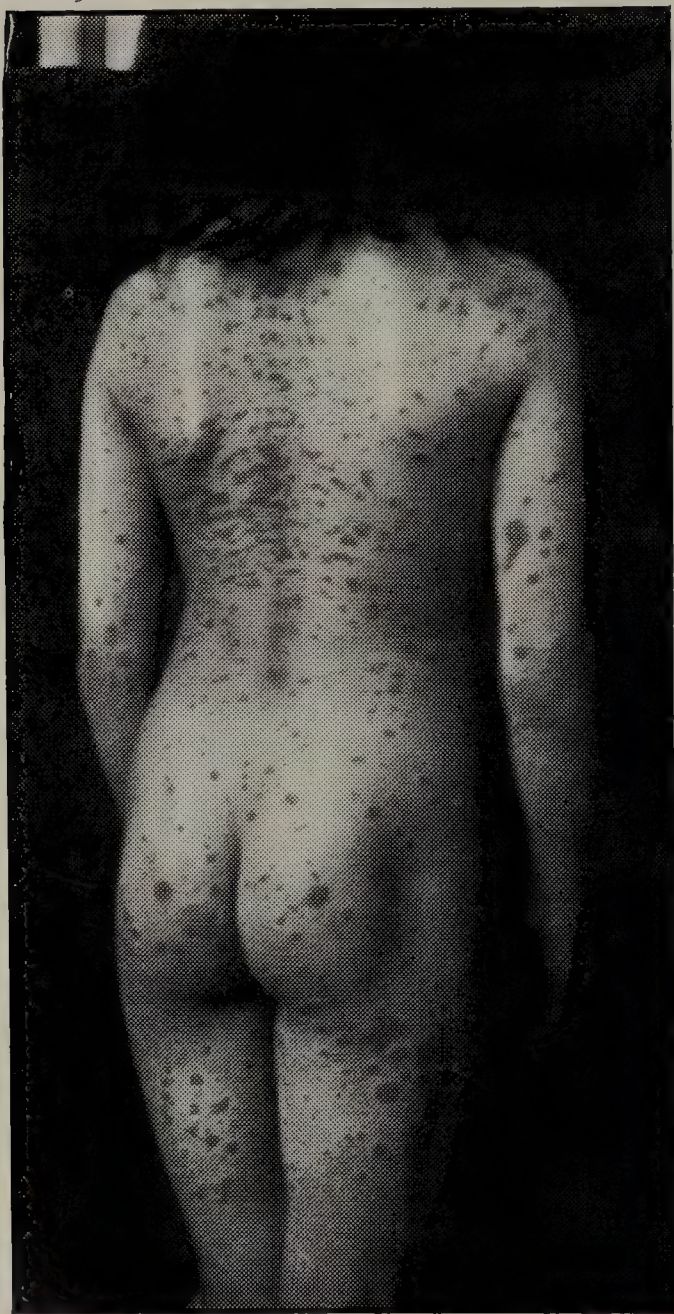


FIG. 121.—Antipyrine Rash.

Drug Eruptions.

Antipyrine Rash.—The rash which is produced in some children by antipyrine is generally seen first on the extensor surface of the elbows and knees and on the thighs and buttocks,

but it may be more widespread in some cases (Fig. 121). It is deep red in colour and papular or morbilliform in type, and in places the spots may run together into blotches. It is sometimes itchy. When such a rash is found, the urine should always be examined for albumin and tube-casts, as the drug is apt to cause an irritation of the kidneys, which, however, subsides on its being discontinued.

Bromide Rashes.—In children who are specially susceptible even small doses of bromide may produce an eruption on the body, face, or limbs and it may persist for months after the drug has been withdrawn. This is generally in the form of acne-like pustules, which may be discrete (Fig. 122) or confluent. In the



FIG. 122.—Bromide Acne in a child of 3 years.

latter type, which is that most frequently seen in babies, we have large rounded or oval raised red patches, the surface of which is studded with yellow spots which represent the centres of the individual acne spots. The rash is occasionally seen in breast babies whose mothers are taking bromide or have been doing so within a few weeks. The administration of arsenic along with the bromide tends to hinder the development of the rash.

Iodide Rashes are much less common than those from bromide, but a discrete eruption of acne-like spots is sometimes met with in children who are taking iodide of iron or some other compound of iodine. Its appearance is sometimes accompanied by a rise of temperature.

Belladonna Rash.—A scarlatiniform rash is sometimes produced in susceptible children by belladonna. It may occur without any extreme dilatation of the pupils, and is unaccompanied by fever.

Serum Rashes.¹

A rash follows the injection of serum in about 10 per cent. of cases in which this therapeutic measure is adopted. The rash, which may be morbilliform, urticarial, or circinate in type, develops at very varying times (four to twenty days) after the injection. There would thus appear to be a definite incubation period for the disease (serum disease). If the child had previously had an injection of serum the appearance of the rash is accelerated; and it should also be noted that if the injection of serum is continued at intervals of less than a week the condition is kept in abeyance.

The urticarial rash is most frequent, the morbilliform next, and the circinate least common. The urticarial may appear as soon as four days after the injection but the circinate not before the eleventh day. The duration of the various rashes is in inverse ratio to their frequency.

Joint pains and fever with enlargement of the glands and œdema sometimes accompany the rash. Constitutional symptoms with cardiac dilatation may also be present, and, as there is not infrequently a leucocytosis, there is a danger of the condition being mistaken for septicæmia.

No treatment is very efficacious in stopping the irritation of the urticarial rash. It may, however, be temporarily relieved by the free application of a carbolic, calamine, and zinc lotion (Appendix E, Form. 49); and the hypodermic injection of epinephrin (0.6 to 1 c.c.) at night is said to give great relief for a short time in some cases.²

Psoriasis.

It is rare for psoriasis to begin before the fourth or fifth year; and in childhood it generally appears first in the form of small, round, scattered spots. The fact of its having begun early in life does not necessarily mean that it will be sure to recur. If the treatment is sufficiently energetic and thorough from the first, it is very likely not to return. In children, as in adults, the key to successful treatment lies in thorough attention being given to the scalp; for it is there that relapses are apt to take their origin.

¹ W. T. G. Davidson, *Glas. Med. Journ.*, 1919, xci., 321.

² Avery, Chickering, Cole, and Dochez, *Monogr. of Rockefeller Inst. for Med. Res.*, 16th Oct. 1917, No. 7, 1.

Urticaria.

Ordinary urticaria is often met with in childhood. It is usually connected with digestive disturbances, though the other symptoms of these may not be very prominent, and it sometimes occurs simply from over-eating. It may, as in adults, be caused by certain articles of diet, and is often associated with asthmatic symptoms (p. 648). Occasionally, it occurs during the active stage of rheumatism. Sometimes urticaria is accompanied by distinct œdema; and when this is so, the eruption may be very slight. In unexplained œdema, therefore, it is always well to inquire particularly as to the presence of itching. Angioneurotic œdema, which is really a form of urticaria, has already been referred to (p. 380). The form of urticaria which is peculiar to childhood is urticaria papulosa, and it is an extremely common affection.

Urticaria Papulosa (*Lichen Urticatus*).

Symptoms.—This condition differs from ordinary urticaria in being a papular or papulo-vesicular affection. The papule begins as the centre of an urticarial wheal, but the wheal rapidly fades. The papule may be topped by a vesicle, and occasionally large bullæ form. The severity of the itching generally leads to the tops of many of the papules being scratched off and replaced by scabs. The eruption is also often complicated by a contagious impetigo spread by the scratching.

The eruption of lichen urticatus is met with chiefly on the extensor surface of the arms and legs, on the loins and buttocks, and on the wrists and hands. The upper part of the face, the genital organs, and the palms and soles are generally free.

The disease is commonest in the summer months and during the first two years of childhood, but it occurs frequently up to the eighth or tenth year. It often lasts for months in spite of all treatment, and it tends to recur. This tendency passes off, however, as the child grows older. It is often attributed to digestive disturbance, but it is generally impossible to be sure that there is any real connection between the two conditions. There is no reason to regard it as an anaphylactic phenomenon (Barber).

The *diagnosis* is generally easy. Occasionally, however,

when it is complicated by impetigo and altered by much scratching, the eruption may resemble scabies; but the characteristic burrows are absent and the distribution of the eruption is quite different. The bullous variety of urticaria papulosa is apt to be mistaken for varicella.

Treatment.—This form of urticaria is often a most obstinate and unsatisfactory malady to treat, as we are so ignorant of its exact causation. The main indications are as follows:—

1. Attend to the digestion and general health, avoid all indigestible articles of diet and *sweets*, and keep the bowels right with magnesia and an occasional dose of grey powder and rhubarb. Sometimes ichthyol in one or two minim doses with glycerine and syrup seems to do good (Appendix E, Form. 13).

2. General sedatives such as antipyrine are often useful in helping the child to sleep. In urticaria papulosa, as in chronic relapsing urticaria, large doses of quinine are sometimes useful. A grain and a half for each year of the child's age may be given at bedtime (Eustace Smith).

3. Local applications almost always give some relief, although often it is only slight and temporary. A 2½-per cent. ointment of β -naphthol may be used or a calamine lotion (Appendix E, Form. 51). Tarry lotions are particularly soothing—*e.g.*, glycerin. plumbi acetat., and liq. picis carbon. āā 2 drm. to 6 oz. water. They should be followed by dusting with starch powder.

Urticaria Pigmentosa (*Xanthelasmaidea*).

Urticaria pigmentosa is a rare disease which is usually seen in early childhood. In a few cases the eruption has been present at birth, but generally it appears during the early weeks or months of life. Cases have been reported in which it only began in adult life.

The eruption is seen mostly on the trunk, neck, and thighs. Sometimes the spots are few in number, but usually they are thickly scattered over the skin in the affected areas. The eruption consists of rounded or oval spots of a peculiar yellowish-brown colour which may be flat (*maculæ*) or slightly raised (*papules*); and it is characteristic of them that they swell and become itchy when exposed to the air or rubbed and also when the child is excited. The general health is quite

unaffected. The diagnosis, which is not difficult, depends on the age of appearance and the distribution of the eruption, its pigmentation, and especially its peculiar urticarial irritability.

The causation and nature of the malady are altogether obscure, but it is regarded now by dermatologists as quite a distinct disease from ordinary urticaria.

Spontaneous recovery takes place about the time of puberty, or soon after, in most cases; and until recently no *treatment* has been found to have any effect upon its continuance. Dr R. Cranston Low states that he has seen great improvement follow the application of X-rays.

Staphylococcal Impetigo.

The form of impetigo caused by *Staphylococcus aureus* differs from other varieties of this eruption in that its lesions occur in and around the hair follicles, and are pustular from the first. It may be set up by dirty applications to the skin, and is a common complication of other skin diseases in children—such as scabies, lichen urticatus, impetigo contagiosa, and ringworm.

Treatment.—Impetigo usually yields rapidly to washing with a weak corrosive solution, boracic dressings, and incision of any boils that form. If the eruption is obstinate, or tends to recur, vaccine treatment should be tried, and is often successful.

Boils (*Furunculosis*).

Common boils are due to the action of *Staphylococcus aureus*. In infants they usually occur as a complication of some cachexia. Their chief peculiarities at this age are their small size, their large number, and the absence of a regular core.

The *treatment* consists in general attention to the nutrition and digestion, including the administration of tonics. Sulphide of calcium ($\frac{1}{4}$ to $\frac{1}{2}$ gr. four or five times a day), and stannoxyl tablets (gr. v., t. i. d.), have been strongly recommended. The skin should be thoroughly purified with bichloride of mercury solution (1 in 2000); and the boils should be incised when they point, their contents squeezed out, and iodoform powder applied. When this is done they heal rapidly in most cases. Far the most effectual treatment, however, consists in the use of a

Staphylococcus aureus vaccine; and this should always be had recourse to if the case is at all obstinate.

Pemphigus Neonatorum.

This consists in a bullous eruption in the new-born due to streptococci conveyed by the hands of the mother or nurse. The mischief may start anywhere and may involve a wide expanse of the body. In severity it varies much. Mild cases respond to treatment with methyl green and gentian violet (āā 5 per cent. in absolute alcohol) applied locally, but the widespread examples are often fatal. In these latter, anti-streptococcus serum may be tried.

Impetigo Contagiosa.

Contagious impetigo is characterised by the formation of vesicles and bullæ which burst almost at once, forming large yellow scabs. These crusts are much larger than those in eczema and there is no itching. When they are picked off, shallow excoriations are left. The disease is extremely common, especially on the face and head. It is due to streptococcal infection and is very contagious. In boys' schools it is rapidly spread by football and such games, and goes by the name of "*Scrum Pox*." Among the poor it is generally associated with the presence of *pediculi capitis*, and when the eruption occurs the hair should be carefully examined for nits (p. 407). In delicate children, if neglected, it may give rise to severe cervical adenitis and even to abscess formation.

Treatment.—The eruption is generally cured quickly by careful removal of the crusts by means of starch and boracic poultices, followed by washing with a weak solution of corrosive sublimate (1 to 4000) and the application of dilute ammonio-chloride of mercury ointment (5 gr. to 1 oz.).

Ecthyma.

In badly-nourished children streptococcal infection is apt to cause deeper skin ulceration and scabbing. It is then called ecthyma.

In this eruption the *treatment* of the child's general health is of great importance. The local treatment is the same as for ordinary impetigo.

Gangrenous Dermatitis (*Varicella Gangrenosa*).

This eruption used to be regarded as a sequel of varicella that was apt to occur in cachectic infants. This is now thought to have been a mistake, due to its early lesions having a superficial resemblance to chicken-pox vesicles. The condition is mostly met with in young babies. Large rounded ulcerations form with a blackened base (Fig. 123), and these spread considerably and run into one another. The patients are generally in an extremely exhausted state, and they rarely recover. The *treatment* consists in the use of stimulants and opiates in



FIG. 123.—Gangrenous Dermatitis.

full doses and in local antiseptic measures. Adamson recommends prolonged soaking in a warm bath, followed by the application of lotio hydrarg. perchlor. (1 in 2000). He says that, under this treatment, the ulcers rapidly heal; and that, if it is carried out early, the child's life may be saved.¹

Exfoliative Dermatitis of New-born Children

(*Ritter's Disease*).

This is an obscure and very fatal disease of the skin which sets in, usually within the first week, or at any rate within the first two or three weeks, in apparently healthy children. The face and the "napkin area" are generally markedly affected, but the whole surface of the skin becomes implicated in time. Flaccid pemphigus-like bullæ occasionally appear and extend

¹ Garrod, Batten, and Thursfield's *Diseases of Children*, London, 1913, 1134.

indefinitely, and by some the condition is believed to be related to pemphigus neonatorum. The adhesion of the epidermis to the underlying parts becomes so loosened that the least touch removes it and leaves the red raw-looking corium exposed (Figs. 124 and 125). Fissures form about the mouth and anus.



FIG. 124.—Exfoliative Dermatitis in a child of 13 days.

There is no rise of temperature, but the child's general strength is profoundly affected and he usually dies within a week in a state of exhaustion. A few cases, however, recover.

The condition is believed to be due to a form of septic infection. It has certainly nothing to do with congenital



FIG. 125.—Exfoliative Dermatitis in a child of 13 days.

syphilis, although it is often mistaken for a manifestation of that disease.

Treatment has generally no effect. Escherich recommends the application of linseed oil and lime-water in the early stages, and Lassar's paste and zinc powder later. Polyvalent anti-streptococcus serum would sometimes seem to do good.

Seborrhœa.

Simple seborrhœa of the scalp in babies is an extremely common condition. It also occurs as dandruff in older children; and, when severe, may be mistaken for ringworm. Its treatment consists in frequent and thorough washing with spirit of soap. In obstinate cases an ointment of sulphur and salicylic acid (āā 15 gr., vaseline 1 oz.) is useful. For the permanent cure of seborrhœic dermatitis on the face, or elsewhere on the body, it is *essential* to get the scalp into a thoroughly healthy condition (p. 386).

Warts.

Ordinary warts are due to an unknown contagium. They come, and also often go, spontaneously in a mysterious way. If they grow from a small base, they should be snipped off with scissors and the base touched with a strong solution of chromic acid (ac. chromic. 1 drm., aq. 45 minims). If they are large, the chromic acid solution may be painted on them once every three days, the burned tissue being sliced off with a knife before each fresh application; or salicylic collodion (1 drm. to 1 oz.) may be applied once daily. When the warts are numerous, a few applications of the X-rays may be tried and will sometimes cause them to disappear rapidly.

Molluscum Contagiosum.

Molluscum contagiosum is a form of wart, due to an unknown infection, which is apt to spread from the common use of towels. When the lesions are few and pedunculated, they may be *treated* by being snipped off. What is even better is to shell out their contents by squeezing between the thumb nails and then to rub carbolic acid thoroughly into their centre by means of a sharpened wooden match. If they are numerous and close together, the X-rays may be used.

Herpes Zoster.

Herpes zoster is commoner between four and twelve years than at any other period of life (Head).¹ Its appearance is generally preceded by malaise and nocturnal restlessness, and

¹ *Allbutt and Rolleston's Syst. of Med.*, London, 1910, vii., 470.

sometimes by vomiting, and there may also be some rise in temperature; but often there are apparently no general symptoms at all. It is characteristic of the disease in childhood that there is little or no pain during the onset; and the occurrence of severe after-pain, which is such a distressing symptom in old people, never occurs in early life. The distribution of the eruption is the same as in adults except that the forehead is very rarely affected. Occasionally it is met with on the mastoid region along with an affection of the tonsil.

During the last few years a great deal of attention has been drawn to the interesting fact that cases which present the typical characters of herpes zoster sometimes infect other children with ordinary varicella.¹ It has also been said that individuals who have suffered from herpes possess an immunity against poliomyelitis.

The *treatment* of herpes zoster consists in the application of zinc dusting powder and a layer of cotton-wool.

Herpes Simplex (*H. Febrilis*).

Herpes febrilis, the eruption of groups of vesicles which so commonly occurs with an ordinary cold, is seen often in pneumonia, cerebro-spinal meningitis, and various other febrile conditions. It is common on the lips, cheeks, and ears. Unlike herpes zoster, it has a strong tendency to recur in the same positions. It is said that "an attack may sometimes be aborted by a few grains of quinine, taken on its first appearance, by rubbing on a strong sulphur ointment, or by painting on collodion. When the vesicles have formed, calamine lotion or zinc ointment may be applied."

Scabies.

The peculiar distribution and the tendency to rapid suppuration of the lesions of scabies in infants lead to its real nature being often overlooked. Scabies does not occur on the face in adults, but the baby's cheeks are apt to be infected from the mother's breasts. In the same way, the mother's fingers

¹ W. R. Brain and E. B. Strauss, *Recent Advances in Neurology*, London, 1930, pp. 283 and 298.

are more likely to infect the baby's feet than his hands. The violent way in which the child's delicate skin reacts to the presence of the parasite and to the scratching it sets up, causes such severe pustulation that it is very difficult in most cases to make out the characteristic burrows—not to speak of finding the acarus. There is invariably, however, violent itching—especially at night; and, when this symptom is present in any case of obscure dermatitis, it is always well to bear scabies in mind. The diagnosis can usually be confirmed by examining the mother's hands. Urticaria papulosa is the disease most often mistaken for scabies.

Treatment.—Provided the application of the treatment is sufficiently thorough, scabies can always be cured in a few days. The child should have a prolonged warm bath and be well rubbed all over with soft soap. After the bath, the ointment should be thoroughly applied and left on for the night. It is to be reapplied every morning and evening for three days. On the fourth day, clean clothes and sheets are substituted for the soiled ones, which are taken away and sterilised by boiling. If sulphur ointment is used, it should be diluted with equal parts of simple ointment. If much irritation of the skin is present, Kaposi's naphthol ointment (Appendix E, Form. 60) may be preferred to a sulphur preparation. Considerable irritation of the skin is often left after the itch is cured; this may require further soothing treatment in the form of zinc and boracic ointment and daily sponging with a tarry lotion (liq. pic. carb. 1 drm., aquæ 1 pint) for a week or so. It is, of course, necessary that all infected persons in the house should be treated at the same time.

Pediculosis.

Pediculus Capitis.—The commonest form of pediculosis in children is that of the scalp. In many cases its presence causes little discomfort. In others there is considerable itching, and it may set up contagious impetigo of the face, scalp, and neck. There are generally few pediculi and many nits, as each female lays from 50 to 100 eggs, and each egg takes about a week to hatch. The diagnosis depends on the recognition of the nits; *and the presence of impetigo on the face should always suggest a search for them* (p. 402).

The hair should be soaked for two nights and a day with

equal parts of paraffin and olive oil. This is washed off with soft soap and hot water on the second morning, and the nits are removed with hot vinegar and a fine tooth comb. Oil of sassafras is also a useful application. It is not necessary to cut the hair short, but doing so saves a great deal of trouble.

Pediculus Corporis.—The body louse is rare in little children. Its main treatment consists in baking the infected clothes, or going over their seams with a hot iron, and then rubbing white precipitate ointment into them.

Pediculus Palpebrarum.—The pediculus pubis is occasionally found on the eyelashes of much neglected children who have been infected from their mother's axilla. The parasites and nits should be removed by forceps, and calomel dusted on, or a weak mercurial ointment applied.

Ringworm.

Ringworm may affect the scalp (*Tinea tonsurans*), the skin of the body (*T. circinata*), or, much less commonly, the nails (*T. unguium*). On the scalp and nails it is usually due to infection from children; but, when the disease is on the body only, it may have come from a dog or cat.

Tinea tonsurans.—Ringworm of the scalp is practically confined to children of school age (seven to fourteen), and those who have been born in India, or are delicate from other reasons, are specially prone to take it. It may be caused by either of two kinds of fungi, microsporon Audouini—the small-spored—and trichophyton megalosporon—the large-spored variety. In Scotland most of the cases are due to the small-spored kind.

In typical cases of the *small-spored variety*, the disease first shows itself on the scalp, as a small, pale, scurfy, partially bald spot. On this, most or all of the hairs are dull and broken over, and their ends are ragged and brush-like. This appearance is so easily seen with a lens, and so characteristic, that microscopic examination is generally unnecessary. When they are examined under the microscope, however, the surface of the broken hairs is seen to be thickly covered with a mosaic of spores.

In other cases, the disease is much more widely spread through the scalp, and diseased hairs are found scattered here and there among the healthy ones. In this, as in all varieties

of ringworm, the diagnosis rests solely on the discovery of diseased hairs.

The *large-spored variety* also produces a rounded, bald spot on the scalp. In one form of it, the hairs are so brittle that they appear like little black dots, and it is difficult to get hold of them with forceps; in another form, the hairs are longer and less brittle and there is little scurf on the patch. On microscopical examination of the large-spored ringworm, the spores are seen in and round the hairs in rows, arranged in a loose network.

When there is difficulty in making sure of the presence of the fungus in any species of ringworm, the hairs should be carefully stained and examined under the microscope. Norman Walker gives the following method as satisfactory: The hairs are first washed in ether to remove the grease, and then steeped for from ten to thirty minutes in a saturated solution of gentian violet in aniline water. They are next transferred for two minutes to Gram's solution of iodine (iod. 1, pot. iod. 2, water 300); then placed on a slide and dried firmly with blotting paper, and a drop of aniline oil, containing enough pure iodine to give it a light mahogany colour, is applied. The fungus will then be visible under a low power.

Kerion.—When Nature attempts to cure the ringworm patch by violent suppuration at the roots of the diseased hairs, we have the condition known as “kerion.” The patch swells, becomes red and boggy, and the hairs fall out. The spontaneous cure, though severe, is unfortunately not usually complete, and sufficient diseased hairs are left to keep up the disease.

Treatment.—As the ringworm fungus penetrates to the bottom of the hair follicles it cannot be satisfactorily reached by parasiticide remedies. The only efficient treatment consists in epilation, and the administration of thallium acetate is now the standard method for this purpose. Accurate dosage of the drug is necessary, otherwise severe toxic symptoms (chorea, gastro-intestinal disturbance, and skin eruptions) develop. Eight milligrams per kilo. body weight (nude) is the most satisfactory dose.

The above procedure has to a great extent displaced the use of X-rays, which are difficult to apply in the proper dosage so as to ensure thorough epilation without causing damage,

although many authorities still consider this method in the hands of an experienced radiologist as the one of choice. The older methods of applying irritants, which were not only uncertain but exceedingly painful, have been entirely given up.

Kerion should be treated by soothing applications such as starch poultices, and by careful epilation on and around the patch.

Tinea circinata.—On the body, ringworm produces somewhat rounded, slightly scaly rings of a pink colour; and in severe cases there may be small vesicles at the periphery (*eczema marginatum*). If a fragment of the epidermis is placed



FIG. 126.—Ringworm of the Nails. (Dr Cranston Low's case.)

on a slide for ten minutes in liquor potassæ, the filaments of the fungus can be clearly seen under a low power.

The treatment of ringworm of the body is a very simple matter compared to that of the scalp variety. Destruction of the superficial epidermis by painting with the strong tincture of iodine, or inunction with ung. hydrarg. ammon. chlor. (B.P.), will cure it in from six to ten days.

Tinea unguium.¹—Ringworm of the nails is an even more troublesome disease than that of the scalp. It is caused by the large-spored fungus, and is very rare in children. In the few cases observed it has not been associated with ringworm elsewhere. Its appearance is quite characteristic (Fig. 126), but it is often mistaken for simple onychia. The fungus can be found in fragments of the nails, if they are steeped overnight in liq. potassæ.

¹ R. Cranston Low, *Edin. Med. Journ.*, Feb. 1911, 121.

The *treatment* by soaking with Fehling's solution which N. Walker recommends is much too painful to be used in children. For them Cranston Low advises scraping with a blunt knife, and rubbing-in iodine ointment.

Favus.

Favus, although much rarer to-day than formerly, is still prevalent in some places in Scotland, and its sulphur-yellow crusts on the scalp with their mousy odour are familiar to most older practitioners. The similar cup-shaped yellow crusts on a reddened base which form on the body are more often overlooked. *Favus unguium* occasionally occurs.

The *treatment* of the disease on the scalp is usually difficult and prolonged. In the very early stages, epilation and the application of an antiseptic ointment may possibly succeed; but, later, nothing short of the X-rays is usually successful.

Favus of the body, which is sometimes due to a different species of the fungus derived from cats or mice, is rapidly cured by removing the crusts and applying a parasiticide ointment.

Alopecia.

In young children irregular patches of baldness on the scalp and eyebrows are characteristic of congenital syphilis, especially when the remaining hair is copious ("syphilitic wig") (Fig. 308, p. 920).

In older children bald patches may result from severe seborrhœa and from ringworm. A diffuse loss of hair may occur after attacks of debilitating illness, especially after erysipelas. The hair usually grows in again rapidly. In cretins a considerable temporary loss of hair is usual during the beginning of thyroid treatment.

Alopecia Areata.—The pale, rounded, bare patches of alopecia areata usually appear suddenly. The skin on them is smooth and often atrophic in the centre, and any broken hairs left are apt to be thinned near the root and shaped like a note of exclamation (!). They are quite unlike those of ringworm. In children, recovery practically always occurs after a number of months, but relapses are not uncommon. In rare cases, the alopecia spreads and becomes general.

Etiology.—The causation is obscure. The disease has been attributed to nervous influence, to the action of bacteria, and to toxins. In some cases it has proved to be contagious and large epidemics have been observed; but, in the great majority of instances, it does not spread.

Treatment.—Tonics and slightly irritating antiseptic lotions and ointments are indicated. N. Walker recommends lactic acid (Appendix E, Form. 7), sulphur ointment, and a lotion of hydrarg. perchlor. in spirit ($\frac{1}{2}$ to 2 per cent.). Ammonia lotions (Appendix E, Form. 8) are also useful. It is advisable to change the application from time to time.

In school children, it is well to insist on careful precautions being taken against the common use of caps, towels, etc., and close personal contact; but the rigid restrictions necessary in the case of ringworm are generally not required.

Diseases and Deformities of the Nails.

The nails are not very frequently affected, but their diseases are often of great interest, and are sometimes helpful because they throw light on disease or deformity present elsewhere.

Onychia (inflammation of the matrix of the nail) may be due to various pyogenic organisms, to tuberculosis, or to congenital syphilis (Fig. 309, p. 921).

Mycosis of the Nails may be caused by favus, or more commonly by ringworm (Fig. 126).

Hæmorrhage into the substance of the nails is occasionally seen in purpura.

Longitudinal Ridging takes place in the nails of fingers which have been persistently sucked for years.

Transverse Ridges sometimes mark the previous occurrence of severe acute illness, such as scarlet fever, enteric fever, or pneumonia. (See transverse marking of bones, p. 257.)

Exaggeration of the Curve of the nails is seen in dwarfing of the terminal phalanges, and, generally, in clubbing of the finger-ends from congenital malformation of the heart and other causes.

The appearance and significance of *bitten nails* have already been referred to (Fig. 55, p. 90).

Thickening of the Nails is sometimes present as a local non-inflammatory disease of obscure origin and of no general

significance; sometimes it is met with in congenital syphilis and in psoriasis.

Defective Development and Growth of the Nails are sometimes seen on the ill-formed fingers and toes of mongols and other mentally defective children. A rare congenital, hereditary, and family condition is occasionally met with (p. 890), in which one parent and several members of a family show marked arrest of development of the nails along with either absence or extreme smallness of the patellæ.

CHAPTER XVII

THE TEMPERATURE

Temperature in Health

Taking the Temperature.—In older children, as well as in babies, the temperature is best taken in the rectum; but, for ordinary purposes, it is more convenient to use the axilla, or in young children the groin, which is better in them because it can be reached with less undressing, and the child is more willing to sit still with the thigh bent on the abdomen than with the arm held close to the side. The thermometer should not be trusted in the mouth in children under four years old, and, even then, only when the child is intelligent and not agitated. When the temperature is taken in the rectum, the thermometer should be oiled before insertion and should be held in place for three or four minutes. When the groin or axilla is used, care must be taken that the skin is dry. In either the skin or groin, an ordinary thermometer should remain about ten, and a “half-minute” thermometer about five minutes, if complete accuracy is desired. If the skin in either situation is cold to begin with, a longer time may be required. In the mouth, five minutes is needed for an ordinary, and three for a “half-minute” thermometer.

The temperature must always be taken in the same place each time, as the rectal temperature is considerably higher than that in the axilla or groin. According to Demme,¹ the difference between the rectal and axillary temperatures may be from 0.5° to 1.6° F. in normal children, and from 0.9° to 1.9° F. in those who are ill.

Normal Temperature.—At birth, the infant's temperature is a few points above that of the mother. In the course of a few hours, it falls below normal but soon rises again. During infancy and childhood, the normal temperature is

¹ 14^{ten} *Jahresbericht des Jenner'schen Kinderspital in Bern*, 1877, 7.

usually said to be a little higher than in adult life; but, according to Finlayson,¹ the mean temperature is really about the same, only the daily range is greater. The exact contour of the daily curve varies somewhat with the age of the child,² as well as with his times of sleep, the amount of his activity, and other circumstances.

Subnormal Temperature.—During the first three or four months of life there is a strong tendency for the temperature to fall very low. In premature and atrophied babies, it often remains constantly subnormal—97° to 95° F., or lower. When this condition is present, a rise to 98° or 99° F., may indicate the presence of fever. After any period of pyrexia in childhood it is usual, and a good sign, for the temperature to remain subnormal for some days.

A prolonged subnormal temperature in a young baby, when accompanied by other signs of ill-health, is a disquieting sign, though not always of serious significance. It may depend on a defect in the diet; too little carbohydrate being given, and withdrawal of salts from the food, are both apt to cause it.

Pyrexia.

Young children are more prone than adults to pyrexia from slight causes, and any cause generally produces in them a higher degree of fever. This may be partly accounted for by the greater dryness of the child's skin in feverish conditions. Emotional causes are also apt to raise the temperature, which probably accounts for the slight rise which normal children sometimes show during the first night they spend in a hospital ward; and in some children muscular exertion does so also.

Rapid rise of temperature from trivial causes are so common in childhood that pyrexia alone is not a sufficient reason for anxiety. Generally it is only when it continues for some time, or is accompanied by other disturbances, that a high temperature becomes a serious symptom.

Sudden Rises of Temperature.—When the temperature of a healthy child rises suddenly and keeps up, the onset of

¹ *Keating's Cyclopædia of the Diseases of Children*, Art. "Diagnosis," 1889, 98; and *Glasgow Med. Journ.*, Feb. 1869, 186.

² Jundell, *Jahrb. f. Kinderheilk.*, May 1904, lxxix., 521.

one of the exanthemata or of influenza may be suspected. Careful and repeated examination should be made for signs of pneumonia. Inspection of the throat is *never* to be omitted ; and otitis and pyelo-nephritis must also be remembered as common causes of fever. In many cases the pyrexia depends on a passing disorder of the digestion, in which case an emetic or aperient often acts as a rapid antipyretic. Erysipelas and cerebro-spinal meningitis are other occasional causes of high fever, and the possible presence of a surgical lesion such as acute osteomyelitis is not to be forgotten.

Dehydration Fever in New-Born Children (*Inanition Fever*).—Many years ago L. E. Holt¹ drew attention to a type of pyrexia frequently seen in breast-babies between the second and fifth days of life which he called “Inanition Fever.” In these cases, when slight in degree, there are no special symptoms beyond fever, thirst, and loss of weight. In those that are more severe, the infant may become collapsed, listless, and apathetic, and look very ill. Occasionally convulsions occur, and the loss of weight is considerable.

The treatment recommended, which is rapidly successful in bringing down the temperature, consists in giving an extra ounce of water every two hours ; and, if this fails, putting the child to a wet nurse, or on the bottle.

It was suggested by J. Comby,² that the fever and general disturbance were due to the infants getting too little milk from the breast, and therefore suffering from local irritation of the internal urinary passages, from the consequently concentrated urine. The resemblance which the severer cases present to those of older babies in the early stage of acute pyelitis, and those with fever from concentrated acid urine, and also the rapidity with which the administration of fluids brings down the temperature, seem to be strongly in favour of this hypothesis. It has also been suggested, however, that the fever in these cases may be due to the absorption of some protein products, bacterial or otherwise, from the intestine.³

¹ L. E. Holt, “Inanition Fever in the Newly Born,” *Arch. Ped.*, Aug. 1895, xii., 561 ; see also H. Bakwin, “Dehydration in New Borns,” *Amer. Journ. Child. Dis.*, Dec. 1922, xxiv., 508.

² J. Comby, *Arch. de Méd. des Enfants*, Oct. 1899, ii., 580 ; also remarks by Christopher and A. Jacobi on Dr Holt’s paper (*loc. cit.*).

³ Grulee and Bonar, *Amer. Journ. Dis. Child.*, July 1921, xxii., 44.

Occasionally we meet with a similar condition of dehydration fever in babies of a few weeks old who are suffering from any cause from lack of fluid, and passing concentrated urine containing uric acid crystals and sometimes albumin. The child's condition may appear serious at first sight, but the administration of copious draughts of water, and alkalinisation of the urine with citrate of potash, are followed by a rapid fall of the temperature and disappearance of the other symptoms (Fig. 127).

Persistent and Recurrent Rises of Temperature.—When there is persistent

or recurrent remittent fever without any ascertainable cause, the diagnosis is often very perplexing. Such a condition may, of course, be due to unrecognised tuberculosis in the peritoneum or elsewhere, or to malaria, influenza, or enteric fever. Some cases of erythema nodosum also have for a time a temperature very like that of mild enteric. Occasionally children who have lived on the Mediterranean or in India, and been fed on goat's milk, show the peculiar long-continued irregular type of fever which is characteristic of Undulant or Malta Fever. This is well shown in Fig. 128. A similar condition is occasionally caused in this country by the *Bacillus abortus*, which is closely related to the organism causing Malta fever.¹

In obscure persistent fever, the condition of the mouth, throat, and ears must *always* be investigated. Even slight stomatitis and tonsillitis are capable of causing a considerable degree of pyrexia; and occasionally the removal of a bad tooth with abscess of gum puts an end to a prolonged fever. In any case of unexplained fever the teeth and gums should be carefully scrutinised. The microscopical examination of the urine also should never be neglected, especially in babies, for in them both pyelitis and nephritis may be responsible for a high remittent temperature (p. 435); and it is to be remembered that the pyrexia sometimes occurs some days before pus appears in the urine. Unrecognised patches of broncho-pneumonia, and

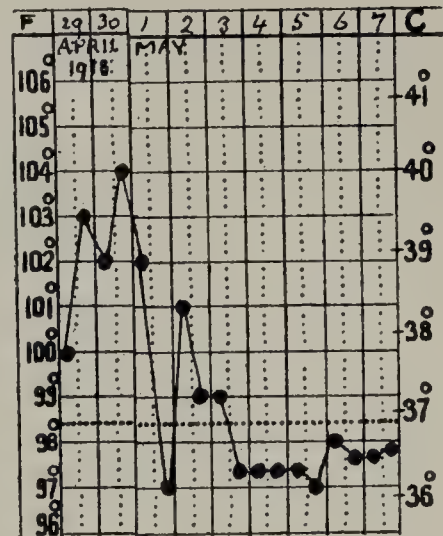


FIG. 127.—Pyrexia from concentrated urine, with uric acid crystals. (Boy of 4 weeks.)

¹ Parker Dooley, *Arch. Dis. Child.*, 1931, vi., 235.

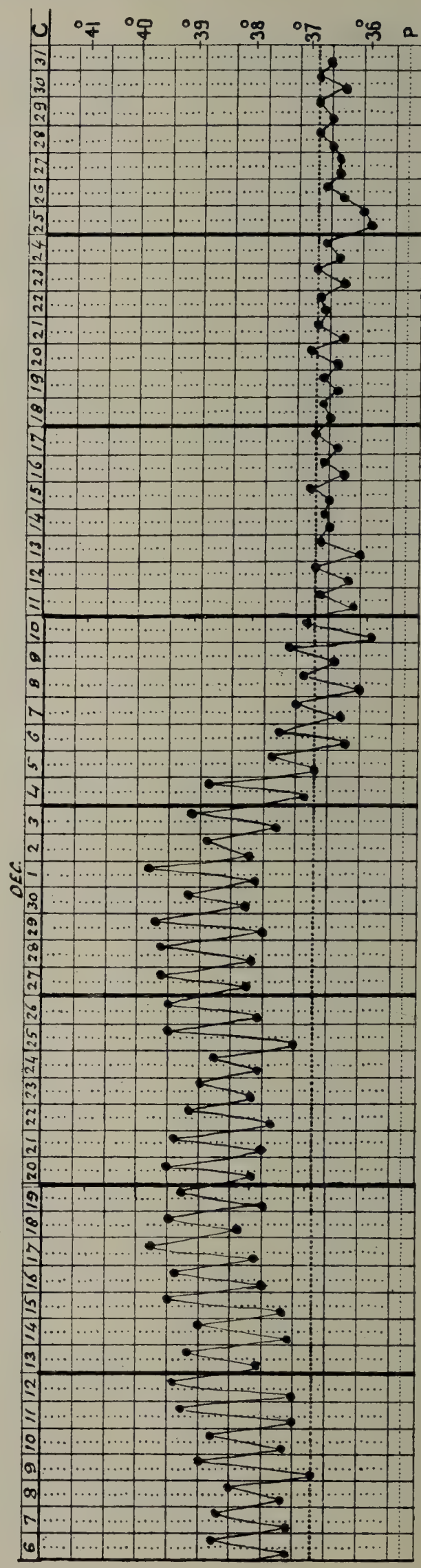
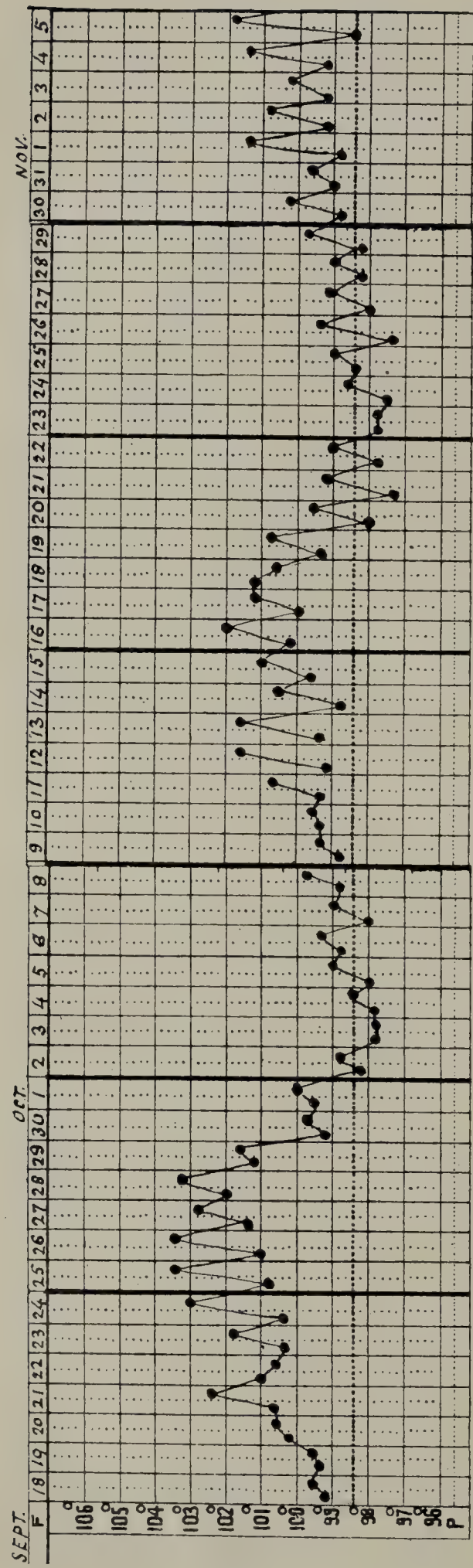


FIG. 128.—Undulant or Malta Fever. (Girl of 5 years.)

in rheumatic children endocarditis and myocarditis, are also occasional causes of fever. Whooping-cough in its early stages is another common cause. Pyrexia is a characteristic symptom of alimentary poisoning. Fever may also be caused by the presence of a round worm in the bowel. The recurrent attacks of fever which are sometimes associated with acetonæmia are referred to elsewhere (p. 558).

Some forms of pyrexia seem to be of a purely nervous origin.

When a child's temperature has for any reason been high for some time, it may continue rising some degrees above the normal for weeks after the original cause has apparently gone. For example, after severe attacks of whooping-cough, it is not uncommon to find a considerable evening rise lasting for many weeks. This may give rise to unnecessary anxiety by suggesting the possible presence of tuberculosis and the child's convalescence be unnecessarily delayed.

A much more serious type of such pyrexia occasionally follows an acute attack of influenza in a young child (see Figs. 129 and 130). The temperature instead of falling to normal in a few days, as it has done in the other children of the family, continues for weeks to exhibit a high remittent type although there are no indications of disease in any of the organs. The pyrexia seems the only symptom, and the child sleeps well, looks well, eats well, and has normal motions. The fever seems, sometimes at least, to depend on a severe derangement of the heat-regulating apparatus, rather than on a continued infective process. The majority of the cases last only a few weeks and end in recovery. They cannot, however, be regarded without anxiety, for weeks of severe pyrexia lower the child's resistance to disease to a dangerous extent, and, after a time, there is a great risk of an intercurrent attack of pneumonia or nephritis. Fig. 129 shows the temperature curve of a previously healthy baby of eight months with this condition, who was treated without any special amount of fresh air, and who ultimately died from broncho-pneumonia and nephritis of a few days' duration. No tuberculosis was found post-mortem. Fig. 130 is the chart of another child of eighteen months whose case was apparently very similar to the last. The child gradually improved on being treated largely in the open air, and ultimately made a good recovery. It is possible, however, that this case may have been one of latent tuberculosis. The elimination of

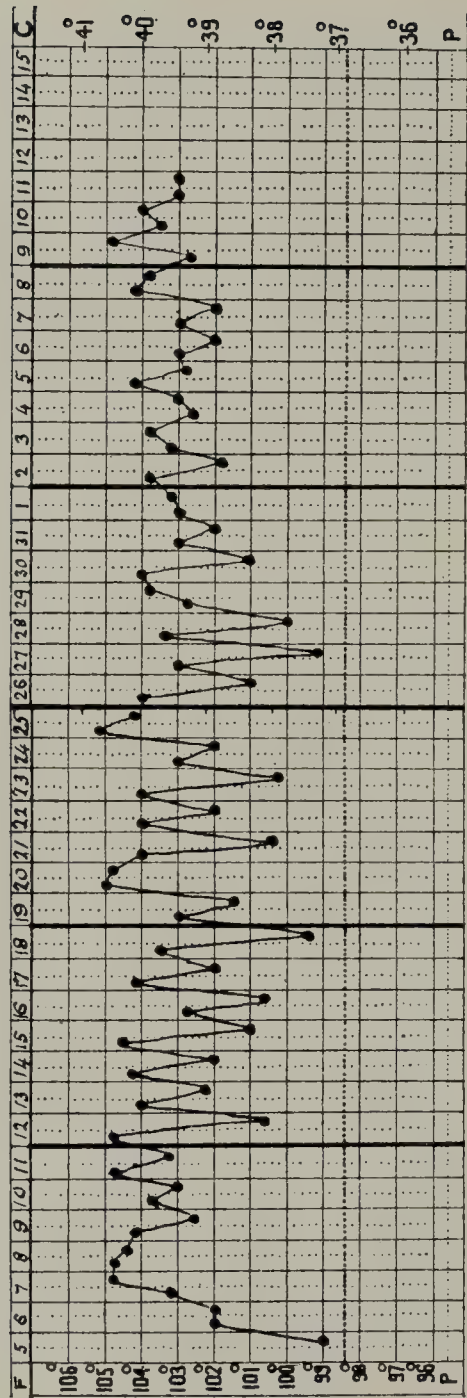


FIG. 129.—Prolonged Influenzal Pyrexia, ending fatally. (Girl of 8 months.)

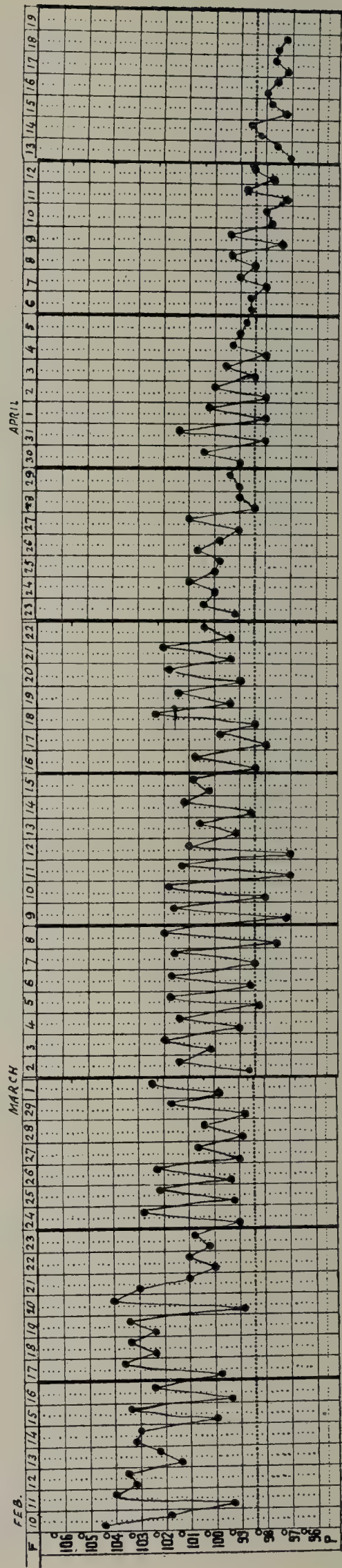


FIG. 130.—Prolonged Influenzal Pyrexia: recovery under open-air treatment. (Girl of 18 months.)

tuberculosis can always be done by employing the Mantoux tuberculin test (p. 947).

Finkelstein¹ has found that a large number of scarlet fever convalescents also show—night and morning—a rise of 1° or 2° F. above the normal. This sets in three or four weeks after the original fever has subsided, and lasts for many weeks. He regards the condition as a nervous disturbance of the heat-centre; and has confirmed this view by observing the effect of intravenous injections of adrenalin. The dose used was from 4 to 7 decimilligrams ($\frac{6}{1000}$ to $\frac{9}{1000}$ gr.) of the German preparation epinephrin, according to the child's age. He found that, after one such injection, the temperature remained normal for two or three days at least, and that in some cases it did not rise again. The effect of the adrenalin could not be relied on, however, if it was given by the mouth or hypodermically. The same injections were found to have no corresponding effect in cases with a rise of temperature due to infection of any kind. Even when the injections fail, therefore, in stopping the rise of temperature in cases of nervous origin, they may still be useful in confirming the diagnosis and dispelling needless anxiety as to the presence of tuberculous or other infection.

In all cases of prolonged pyrexia without apparent cause the *free use of open-air treatment* is strongly indicated. Antipyretics have rarely any effect.

Hyperpyrexia.—It is not very rare in babies to meet with hyperpyrexia. It occurs in various diseases, but is oftenest found in cases of severe intestinal derangement.² Its occurrence in them is ascribed to absorption of the toxins of non-pathogenic organisms taken in decomposing food.³ When it occurs in such cases it is a sign of great danger, as convulsions and coma are apt to ensue, and it calls for prompt and active treatment. The stomach should be washed out and a dose of calomel or castor oil given. All fermentable food, and especially all forms of milk, must be withheld for a time. Stimulants and subcutaneous saline injections should be freely given and cold applied to reduce the temperature. Opiates of any kind must be avoided.

¹ *Journ. Amer. Med. Assoc.*, 23rd Aug. 1924, lxxxiii., 611.

² Chapin, *Arch. of Pediat.*, Nov. 1895, 917; Milton Miller, *ibid.*, May 1897, 347.

³ Centanni, *Deutsch. Med. Wochenschr.*, 22nd Feb. 1894, xx., 176.

In very young and feeble infants the injudicious application of artificial heat (*e.g.*, from hot-water bottles) may cause an alarming rise of temperature. Holt mentions one case in which the temperature rose to 107° F. from this cause. It may be mentioned that rheumatic hyperpyrexia is unknown in young children and is very rare even in older ones. Hyperpyrexia during childhood is probably most frequently encountered during the course of meningitis.

Rigors.

The occurrence of rigors at the onset of fever is rare in childhood, and especially so in early infancy. Their place seems sometimes to be taken by convulsions, but it is certain that this does not occur so commonly as is usually taught.¹ Vomiting is really the common early symptom of disease in infancy and childhood. In older children rigors may be a symptom of many various conditions—such as scarlet fever, enteric, malaria, and pneumonia, and are especially met with in acute septic bone disease and inflammations of the urinary tract.

When rigors occur in infants under two years, they have considerable diagnostic importance, because they are almost always due to the presence of acute pyelo-nephritis (p. 435). We have met with severe rigors in girls as young as five and seven months who had this disease.

¹ Baldwin, "Rigors in Children," *Lancet*, 1896, i., 1635.

CHAPTER XVIII

THE URINARY SYSTEM

IN the clinical examination of children few things are more important than the investigation of the urinary tract. Its maladies are not uncommon; they are frequently serious; and they are very easily overlooked. This is especially true in early infancy, because then even acute diseases of these parts may exist without giving rise to any ascertainable localising symptoms, apart from the state of the urine. A knowledge of its condition is also often essential for prognosis as well as for diagnosis. Even in the matter of treatment it is not without importance, for there are many drugs which should never be given in full doses until we have ascertained that the urine is fairly normal in amount as well as in character.

The Urine.

Its Collection.—In infants, although difficult and troublesome, it is always possible to collect the urine for examination. Its amount, colour, and odour may be judged of, roughly, from the napkins, and we may find on them crystals of uric acid which look like grains of red sand. In all cases, however, it is extremely important, if possible, to obtain a proper specimen for microscopical as well as chemical examination. There are various ways of getting this. One plan—which is the least satisfactory—is to leave in contact with the genital organs a piece of absorbent cotton wool which can be afterwards squeezed out into a glass. A simple device—which sometimes succeeds—consists in getting the nurse to waken the child from sleep and at the same time to exert steady pressure over the bladder. In the case of boys, a test-tube or bottle may be so fixed that any urine passed will find its way into it; and in that of little girls a padded bed-pan may sometimes be made use of successfully. If other means

fail, or if the specimen is required for bacteriological purposes a soft catheter should be passed.

Quantity. — The daily amount of the urine varies considerably in different children, and also on different days according to the quantity of fluid taken and the amount got rid of by the skin and bowel. The following Table (Holt) is a summary of previously published estimates.

Average Daily Quantity of Urine in Health.

Age.	Grammes.	Ounces.
First twenty-four hours . . .	0 to 60	0 to 2
Second twenty-four hours . . .	10 „ 90	$\frac{1}{3}$ „ 3
Three to six days . . .	90 „ 250	3 „ 8
Seven days to two months . . .	150 „ 400	5 „ 13
Two to six months . . .	210 „ 500	7 „ 16
Six months to two years . . .	250 „ 600	8 „ 20
Two to five years . . .	500 „ 800	16 „ 26
Five to eight years . . .	600 „ 1200	20 „ 40
Eight to fourteen years . . .	1000 „ 1500	32 „ 48

These estimations for infancy are supported by Rougichitch¹ who, working in R.H.S.C., Glasgow, found the daily output in healthy bottle-fed infants to rise from 10½ oz. at six weeks to 20 oz. at one year. Occasionally larger amounts are passed. Goodhart and Still,² however, found the daily amount of urine passed by children over two years to be much less than is represented by these figures. They say that, for practical purposes, the average daily amount in ounces, between three and twelve years, may be arrived at with sufficient accuracy by multiplying the child's age by two and a half. Similar small amounts have been observed by Churchill and Kerley.

A *diminution in the quantity* of urine passed is often important as an indication that the baby is not taking enough milk, or not retaining it, as in the case of severe vomiting. Scanty urine is also frequently seen during the onset of serous effusions into the pleura and peritoneum, and in cases of profuse diarrhoea.

Increase in the amount passed is seen during the absorption of pleural and peritoneal effusions and œdema. *Polyuria* may

¹ O. S. Rougichitch, *Arch. Dis. Child.*, 1926, i., 289.

² *Diseases of Children*, 11th edit., London, 1921, 537.

be a manifestation of chronic interstitial nephritis (p. 448), of hysteria, and of cerebral tumour (symptomatic diabetes insipidus). It may be an accompaniment of, and indeed play some part in the cause of, nocturnal enuresis. Polyuria is specially characteristic of diabetes mellitus (p. 526) and is often the symptom for which advice is sought. It is, of course, the dominant feature of **diabetes insipidus**, an idiopathic affection which is characterised by the passage of large quantities of normal urine of low specific gravity. This condition is not uncommon in children; it may be congenital, and occasionally shows an hereditary tendency. The cause is ascribed to some vasomotor disturbance of the renal vessels. Associated with the polyuria is polydipsia, but otherwise there may be no symptoms of disease, and, with the exception of a low specific gravity, nothing abnormal is detected in the urine. Occasionally, however, it may induce diurnal or nocturnal enuresis. Nutrition is often well maintained. The prognosis, so far as life is concerned, is good: the condition may undergo spontaneous cure, but, on the other hand, it may persist throughout life.

The *treatment* consists mainly in attention to the general health. Care must be taken to avoid chills and over-exertion, and to encourage the action of the skin by warm clothing and hot baths. To what extent benefit is derived from medicines is doubtful. The subcutaneous injection of pituitary extract has sometimes been found to reduce the quantity of urine considerably. Cod-liver oil, fresh air, and sunshine are indicated. Improvement has also sometimes followed the use of ergot and antipyrine.

In one patient, a healthy-looking boy of two years, whose symptoms had lasted for several weeks, and who was passing from 7 to 8 pints of urine daily, the continued administration of ergot was accompanied by gradual improvement. Two years later, he was reported to be quite well in every respect.

In another case, a girl of twelve years, after many lines of treatment had been tried with no result and the child was in a dangerously weak state, apparently spontaneous recovery took place, and twenty years later there had been no return of the symptoms.

Specific Gravity.—In young infants after the first two days of life the sp. gr. is often very low (1002 to 1004); in

older children it is usually, under normal conditions, from 1010 to 1035.

Reaction.—In children the urine is generally faintly acid. In young infants it often has, however, a strong tendency to turn alkaline soon after it is passed. A high degree of either acidity or alkalinity generally signifies some digestive or other disorder.

Urea.—The daily amount of urea passed varies, of course, with the intake of protein. On a general diet it amounts, in children from three to five years old, to 13 or 14 grammes, and in those from five to thirteen years, to from 16 to 21 grammes (Holt). Campbell, working in R.H.S.C., Glasgow, found the average daily excretion of urea on a diet of 100 c.c. of milk per kilo body weight, to vary in the healthy child from 0.8 to 1.0 gramme per kilo body weight, and the average daily concentration in the 24 hours sample of urine to range between 1.2 and 1.8 per cent.

Uric Acid.—The proportion of uric acid in the urine is much greater during the first eight or ten days than at any later period, and the kidneys often show an accumulation of crystals (uric acid infarcts) in the straight tubules. These crystals are usually washed out within the first week or two of life, and may be seen on the infant's napkins; their passage is, at times, a cause of dysuria, albuminuria, and hæmaturia.

Phosphaturia.—Large quantities of triple phosphate crystals are sometimes found in children's urine. This may cause enuresis. We have seen it accompanied by severe dysuria in a little girl of four years old. Such symptoms are to be treated by acid phosphate of soda (5 to 10 gr., thrice daily), and an increase of meat in the diet.

Ammoniacal Napkins.—About the age of teething, the wet napkins and other underclothes of some otherwise healthy little children emit from time to time a strongly pungent odour of pure ammonia, quite different from the unpleasant ammoniacal smell of decomposing urine. Yet the urine acquires no such odour when passed into, and left standing in, a vessel. The ammonia is of course derived from the ammonium compounds in the urine, which are more plentiful at this age than earlier, but their decomposition in this way is not yet quite thoroughly understood.

It has been attributed to some dietetic error increasing the

ammonia or urea in the urine; but this does not explain why it should only occur when the urine is in contact with the diaper and not in a vessel. Some have suggested, with more probability, that it is caused by an unduly alkaline state of the napkins, such as may easily arise from their having been washed with strongly alkaline soap or soda, and not thoroughly rinsed afterwards in clean water, or from the presence of alkaline stools on the diapers.¹ The presence of a special organism in the napkins which produces ammonia by fermenting the urine has also been demonstrated.²

In some cases the ammoniacal urine seems to do no harm, in others it sets up a painful dermatitis in the napkin area and other parts of the skin which are in close contact with the wet diapers; this may go the length of producing blisters. In boys the end of the prepuce and the point of the penis are often severely affected; and, especially if the child has been circumcised, an obstinate ulcer may form round the meatus, which causes dysuria and sometimes incontinence and, during the process of healing, definite stenosis.³

The irritation of the skin certainly often comes to an end rapidly when the napkins are carefully washed; and, in other cases, when this does not succeed in stopping it, it ceases when the diapers are regularly sterilised by boiling or by soaking them in a solution of corrosive sublimate. When the source of the irritation is removed, the ulcer round the meatus usually heals rapidly if it is kept moistened with boric ointment.

Albuminuria.—In new-born children, during the first week or ten days, a degree of albuminuria is extremely common. Finkelstein⁴ ranks it along with the usual loss of weight which occurs at the same time, and with icterus neonatorum, as a purely physiological phenomenon.

The frequent occurrence of albuminuria under many conditions, apart from nephritis or pyuria, is characteristic of childhood, and its presence is often of little importance. Generally, the finding of a small amount of albumin need cause no anxiety, provided the child seems well and there are none but hyaline casts, a normal specific gravity, and no cardiac

¹ Zahorsky, *Amer. Journ. Dis. Child.*, 1915, x., 436.

² J. V. Cooke, *ibid.*, Nov. 1921, xxii., 481.

³ J. Brenneman, *Trans. Amer. Pediat. Soc.*, 1920, xxxii., 52.

⁴ *Lehrb. d. Säuglingskrankheiten*, 2^{te} Aufl., 1921, 177.

enlargement. Albuminuria is sometimes set up by the application of tarry preparations or carbolic acid to the skin, and occasionally by the internal use of such drugs as chlorate of potash, antipyrine, salicylate of soda, or arsenic in large doses. It is also frequently present in some forms of dyspepsia and in food intoxication; and, in older children, it may occasionally follow habitual over-indulgence in butcher meat. As has been already mentioned, albumin is often found in the urine of new-born children along with the passage of uric acid crystals; and a similar occurrence in older children is a not uncommon symptom of oxaluria. Albuminuria in schoolboys, after foot-races and other forms of violent exercise, is a normal occurrence (physiological albuminuria).

Postural, Orthostatic, Lordotic or Cyclical Albuminuria.—

Unless the possibility of this variety of albuminuria be always kept in mind errors in diagnosis will often arise, and the parents be caused much unnecessary anxiety. It is a functional condition and only appears when the child is in the erect posture; hence the terms *postural* or *orthostatic albuminuria*. The first urine passed immediately on rising contains no albumin, but within an hour or two a considerable amount may be present. This increases during the morning and then diminishes in amount later in the day, and has usually disappeared by the late afternoon and evening. It is on this account that the term *cyclical* was first suggested by Pavy.¹ It has been said that, in contrast to albuminuria due to organic disease of the kidney, the larger proportion is of the nature of serum globulin, but this is certainly not an invariable finding. The urine is usually devoid of all cellular elements, though occasionally a few hyaline casts may be present. Blood is never present, as also any other evidence of renal disease. In fact, excepting for the presence of albumin in the urine at certain periods of the day, which has often been discovered in the course of a routine examination, the child is usually perfectly well.

The condition is seldom observed in children under school age. It becomes tolerably frequent about puberty and is present in something like 30 per cent. of adolescents. Most observers are agreed that it is met with more frequently in girls than in boys.

¹ Pavy, *Lancet*, 1885, ii., 706.

The pathogenesis of the condition is still obscure. Histological examination of the kidneys has never revealed evidence of disease.¹ Jehle² has attributed it to congestion of the kidneys, either from gravity alone or through kinking of the renal vessels induced by lordosis, hence spoken of as *lordotic* albuminuria. Lordosis artificially induced usually brings it about in patients subject to it and hence is a valuable clinical test.

Some writers hold that posture alone is not enough to account for the condition. Erlinger and Hooker,³ Politzer, and Noeggerath and Nitschke¹ believe that there is an *angio-neurotic* element of the renal vessels in the condition. Various functional abnormalities of the circulation have been described in children with this complaint: Gotzky,⁴ for example, found well-marked dermatographia in his cases and Noeggerath mentions its association with "school anæmia" (p. 473).

As previously mentioned, there are usually no symptoms of ill-health but there may be other angio-neurotic manifestations. Sometimes the children are hypotonic.

The diagnosis of this type of albuminuria turns on the absence of all evidence of renal disease—*e.g.*, casts in the urine, high blood urea, high blood pressure and impaired renal efficiency—and the absence of albumin so long as the patient is in the recumbent position. The following test should always be carried out if albuminuria of this type is suspected. Immediately after the bladder is emptied the child is given a drink of water and made to lie down for twenty minutes. The bladder is again emptied, and he is then made to stand in a posture with extreme lordosis for a further period of twenty minutes, when urine is again passed. If the condition is orthostatic albuminuria, albumin will be present in the first and last specimens, but absent from that passed after lying down.

As the condition is physiological no treatment is called for.

Hæmoglobinuria.—Paroxysmal hæmoglobinuria is not very rare in children, and the cases do not differ much from those

¹ C. Noeggerath and A. Nitschke, *Handbuch d. Kinderheil.* (Pfaundler and Schlossmann), Berlin, 4th edit., 1931, Bd. iv., 54.

² L. Jehle, *Die lordotische Albuminurie, ihr Wesen und ihre Therapie*, Leipzig, 1909.

³ J. Erlinger and D. R. Hooker, *Johns Hopkins Hosp. Report*, 1904, xii., 145.

⁴ F. Gotzky, *Jahrbuch. f. Kinderheil.*, 1910, lxxi., 427.

in adults. The patients are usually the subjects of congenital syphilis. Hæmoglobinuria may be observed after blood transfusion if the donor's blood were not of the correct group, and it is sometimes met with as a concomitant of Raynaud's disease.

Coloured Urine.—A dark *sage-green* colour of the urine is found, along with blood corpuscles, albumin, and casts, in carbolic acid poisoning, either from absorption from dressings, or from the internal use of such drugs as salol.

Bright pink urine indicates that the patient has been eating sweets coloured with an aniline dye.

The very rare congenital abnormality known as *Alkaptonuria*¹ is characterised by urine which looks normal when passed, and gradually darkens on standing to a brown and in time to a *black* colour. It reduces Fehling's solution, although it contains no sugar. Many of the cases of alkaptonuria reported occurred in more than one member of a family, and the patients are often the children of first cousins. The disease causes no symptoms, except slight dysuria; but the condition of the urine gives trouble on account of its staining the clothes. It is not amenable to treatment.

Hæmaturia.—This is a common, and generally an important and interesting symptom in childhood. It is met with under a great variety of conditions.

In young babies who are passing uric acid crystals there is often a small amount of blood in the water. It is also very common in infantile scurvy, and its presence in doubtful cases is strongly in favour of the diagnosis of this disease. It is sometimes met with apart from any other characteristic symptom of scurvy. In cases of renal tumour, hæmaturia generally occurs sooner or later. Blood in the urine is also found in many other diseases such as acute nephritis, acute cystitis, purpura, hæmophilia, leukæmia, calculus, genito-urinary tuberculosis, bilharzia, malaria, and other tropical diseases. It is also sometimes caused by the use of cantharides blisters in young children, or by the external as well as internal use of carbolic acid. In moribund babies the sudden occurrence of profuse hæmaturia is often due to thrombosis of one of the renal veins.

¹ A. E. Garrod, "Croonian Lectures," *Lancet*, 1908, ii., 1, 73, 142, and 214; and *Inborn Errors of Development*, London, 1908, 41.

In cases of chronic hæmaturia of obscure origin it is important if possible to have a cystoscopic examination without delay, because if this is not done early in the case of a malignant tumour, the time for the successful removal of the disease may pass before any other symptom develops.

Rarely, a child is met with who has more or less constant hæmaturia, dating from early infancy, without any discoverable cause. These cases are sometimes due to a local abnormality of the blood vessels in the renal pelvis. The children are otherwise healthy, and the hæmaturia is not influenced by any treatment. In one case of this kind the patient grew up fairly healthy in spite of its continuance and served in the army during the War.

Pyuria.—Pus in the urine in girls may be due to a vulvar or vaginal discharge, or in boys to balanitis, and these possible sources must always be investigated. In older children it may, though rarely, be due to renal tuberculosis. Pyuria may be present in infants and yet both naked-eye and histological examination of the whole urinary tract fail to reveal any cause. In the vast majority of cases in infants and children, however, it indicates an infection of the urinary tract. The seat of the mischief may be in the kidney (*suppurative nephritis*) or bladder (*cystitis*), although according to some authorities the pelvis of the kidney (*pyelitis*) or the pelvis of the kidney and the kidney substance (*pyelo-nephritis*) are the common sites.

Although we intend to discuss Pyuria under two main headings — Suppurative Nephritis and Cystitis — it is often impossible to decide either the seat or extent of the mischief, and the simple term “pyuria” is the most applicable. It is also important to remember that the particular infection may have been called into being, or be prevented from healing, more especially in cases of long standing, in consequence of some non-pyogenic affection of the urinary tract. These may be classed as contributory causes. Such are calculus, kinks in the ureters, stenosis of the ureter from aberrant vessels, urethral valves or strictures with dilatation of the bladder, diverticula of the bladder, etc. (p. 449). Thus, although in the first instance, and especially in the more acute examples of recent origin, it is permissible to consider the condition as arising *ab initio*, when response to treatment is delayed, thorough X-ray examination with cystoscopy and catheterisation of the ureters and

pyelography are called for. Smith of Boston¹ and Helmholz² of Rochester have revealed how frequently in the chronic and intractable case of pyuria some such underlying factor is at work.

Bacteriology.—The following Table shows the types of organisms found in the cases of pyuria of infancy and childhood investigated in R.H.S.C., Glasgow, and is in general agreement with the findings usually recorded.

Organism.	No. of Cases.
B. coli (alone)	159
B. coli and streptococci	31
B. coli and staphylococci	1
B. coli and enterococci	3
B. coli (non-lactose fermenting)	1
B. proteus	2
Streptococcus	8
Staphylococcus	3
Total	<hr/> 208

Route of Infection.—As the etiological organism is in the vast majority of instances the *Bacillus coli*, and as it has been shown that the particular strain of coli present in any individual case is the same as that which is present in the bowel, it has generally been concluded that this is the source of the infection. But the route by which it reaches the urinary tract is the subject of much difference of opinion. It has been variously suggested that this may be *via* the urethra, the blood stream, the lymphatics, and also directly through the wall of the bowel to some contiguous part of the urinary tract. There is no doubt that all these methods of spread do occur. Extension directly through the wall or *via* the lymphatics is probably rare, but an appendix abscess, or a loop of ulcerated bowel, may become adherent to the bladder and, with or without the formation of a fistula, lead to a secondary infection of the urinary tract. In these instances faecal contamination of the urine may be present and this possibility should always be borne in mind when investigating a case.

It is, however, either by the blood stream or *via* the urethra that the infection usually spreads, and perhaps the greatest body of opinion inclines to the latter route being, if not the only one, at least the most common. The ease with which the orifice of

¹ R. M. Smith, *Amer. Journ. Dis. Child.*, 1924, xxviii., 678.

² H. F. Helmholz, *Journ. Amer. Med. Assoc.*, 1927, lxxxix., 1932.

the urethra during infancy becomes contaminated with faecal matter is supposed to point to a ready source of infection. The shortness of the urethra in the female, and the fact that girls are more frequently affected than boys, are considered to be in favour of the urethral route. There is no doubt that throughout childhood girls are more commonly attacked than boys—in the proportion of 4 to 1—but in infants under six months, when faecal contamination of the urethral orifice is most common, boys are just as frequently attacked as girls. Of 57 examples under six months of age observed at the R.H.S.C., Glasgow, 54·4 per cent. were boys. The comparative safety of catheterisation in the normal child, the rarity with which cystitis follows urethritis until a stricture has developed, and the efficacy of the modern treatment of gonorrhœal urethritis by auto-lavage, all render this mode of entry of the etiological organism unlikely.

On the other hand, many pædiatricians believe that the infection is of hæmatogenous origin. The fact that the disease, especially in infancy, is apparently often primarily in the kidney, or pelvis of the kidney, would seem to support such a contention. The fact, too, that it is occasionally secondary to tonsillitis and pneumonia, and that the etiological organism is either a streptococcus or the pneumococcus is also in favour of this view. Chown,¹ from a study of post-mortem material at Baltimore, concluded that the "common lesion is a multiple focal, suppurative, interstitial nephritis with which is associated occasionally some degree of pyelitis, and rarely cystitis." Griffin,² from a similar investigation in Glasgow, states that the evidence points to a blood-borne infection in the majority of cases, as the kidney substance was the most severely affected. McLellan, from an analysis of 91 children under our own observation during the years 1925 to 1928, noted that in the cases following gastro-enteritis, renal-casts frequently preceded the appearance of pus and organisms, and that all the five children in whom positive blood cultures were obtained were of this type. This would point to a blood infection. McLellan also noted that a positive blood culture was never obtained in any case in which renal abscesses were discovered post-mortem.

An attempt has been made to decide the question by

¹ B. Chown, *Arch. Dis. Child.*, 1927, ii., 97.

² M. A. Griffin, *Glas. Med. Journ.*, 1930, cxiv., 21.

experiments in animals. Helmholtz has found it impossible to induce infection in animals either by the introduction of organisms into the bladder or by their intravenous injection. From the older work of Guyon, however, one very significant fact emerged.¹ If the bladder is previously injured by cauterisation then the introduction of organisms is followed by infection. In the same way in man, if there is some abnormality of the urinary tract which hinders the normal drainage, *e.g.*, urethral stricture or hydronephrosis, catheterisation becomes one of the most dangerous of procedures. Thus, during pregnancy, from pressure on the ureters or urethra, and in gastro-enteritis following the intravenous injection of hypertonic saline from the diminished urinary output, we find a ready explanation of the frequency of infection of the urinary tract. It may be, too, that it is from toxic action on the kidney and bladder that pyogenic infection of the urinary tract occurs as a complication of febrile diseases like pneumonia. And finally, the possibility of a special susceptibility of the kidney and bladder during the early years of life being another contributory factor should be borne in mind.

SUPPURATIVE NEPHRITIS (*Pyelo-nephritis or Pyelitis*).—It has already been remarked that post-mortem evidence points to the kidney being the seat of the mischief, and that the pelvis of the kidney is frequently devoid of all signs of disease. Hence pyelitis, the usual term, is considered by many pædiatricians a misnomer. Apart altogether from anatomical considerations, it would be remarkable if inflammation of the pelvis of the kidney, being as it is outside the body and non-absorptive, should be accompanied by such severe toxic symptoms as are ascribed to it. When toxic symptoms do occur they are most probably in consequence of mischief in the kidney.

Suppurative nephritis is one of the commonest diseases of infancy and early childhood, and as pyuria or bacilluria is the only definite objective sign it can be easily missed. It is one of the so-called obscure causes of fever, and it may be taken as a golden rule that in any sick infant the urine must be examined. Suppurative nephritis may be primary, when it sets in during apparent good health, but it is frequently a complication of other diseases, especially gastro-enteritis, or it may

¹ *Encyclopedie Francaise d'Urologie*, Paris, 1921, t. iv., p. 186.

follow cystitis. This latter sequence is often suggested by the history of the frequent passage of strongly-smelling urine prior to the onset of the acute symptoms.

The onset is generally sudden, the temperature rising rapidly to 101° or 105° F. and assuming a remittent type like that of enteric fever (see Figs. 131 to 135). The child as a rule has an unusual degree of pallor and is the picture of misery and apparently suffering pain. At other times there is marked drowsiness, occasionally a squint, and thus a meningeal condition is suggested. Very frequently there is anorexia and vomiting with some diarrhoea simulating gastro-enteritis. According to the degree of fever is an increased rate of the respirations, so that some pulmonary infection is imitated. John Thomson stated that in fully one-half of the cases in girls—though scarcely ever in boys—distinct shiverings or well-marked rigors take place. Less commonly, the child may from time to time suddenly become pale, stiff, and collapsed. These faint attacks are very characteristic of the condition. In a few of the cases, especially in young babies, convulsions occur which sometimes seem to begin as rigors. A considerable degree of leucocytosis (16,000 to 32,000) is generally present.

Local symptoms are either trivial or apparently absent and, as previously mentioned, the true nature of the malady would in the majority of cases be overlooked unless a routine examination of the urine were made. There may be, however, colicky pains, some dysuria or frequent micturition, tenderness in one or other loin, and occasionally palpable enlargement of one or both kidneys with pain on pressure, more frequently the left than the right. Enlargement of the kidneys usually signifies abscess formation. In many cases there is definite impairment of renal function. Vulvitis and vaginitis are rarely found.

The urine, when passed, is opalescent and distinctly acid, and contains a considerable amount of pus and colon bacilli, and usually only a trace of albumin. On standing, a deposit is slow to form. Microscopic examination of a drop taken from the middle of a specimen of urine—not from the deposit—shows about six to twelve pus cells in a high-power field. Sometimes in the very early stages of the disease very few or even no pus cells may be present. It is also important to remember that in any specimen of the urine passed at one time

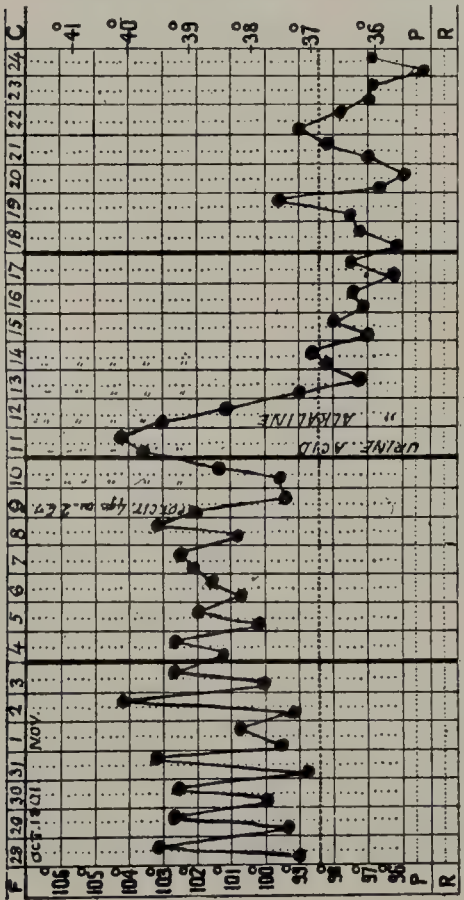


FIG. 131.—Acute Pyelitis, showing effect of alkaline treatment. (Girl of 18 months.)

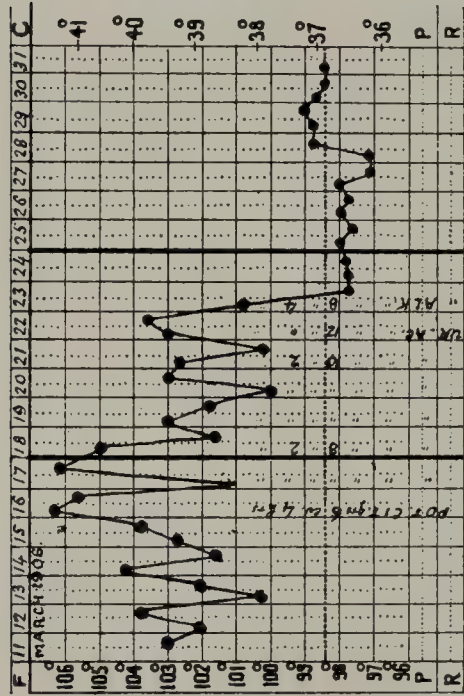


FIG. 132.—Acute Pyelitis, showing effect of alkaline treatment. (Boy of 2½ months.)

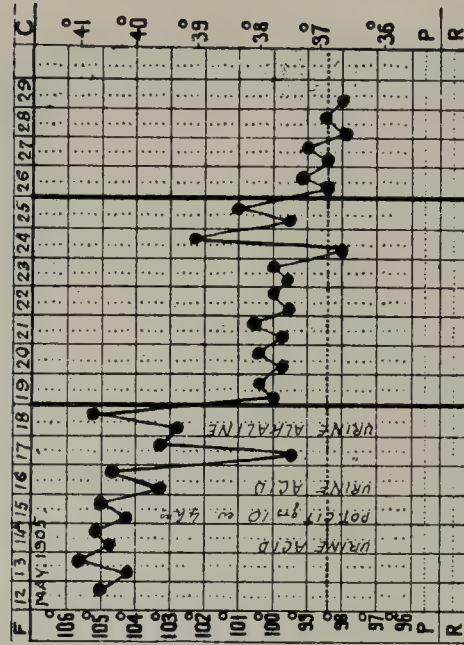


FIG. 133.—Acute Pyelitis, showing effect of alkaline treatment. (Girl of 6 months.)

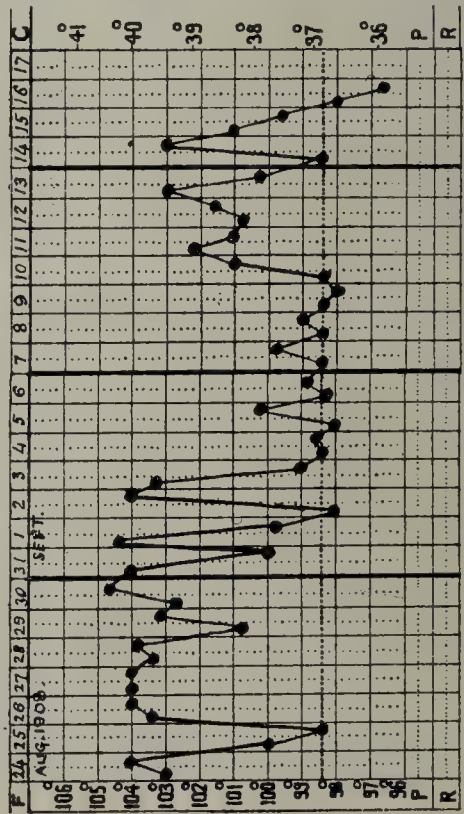


FIG. 134.—Acute Pyelitis, showing effect of alkaline treatment. (Girl of 19 months.)

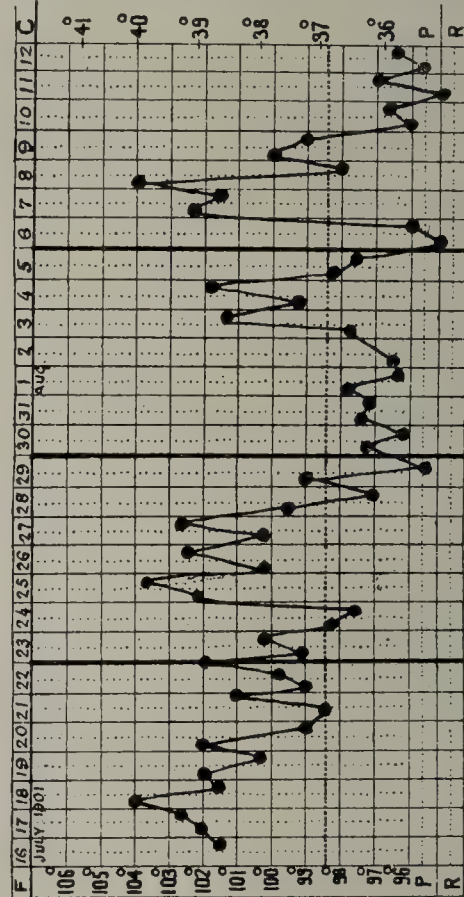


FIG. 135.—Acute Pyelitis, showing effect of alkaline treatment. (Girl of 7 months.)

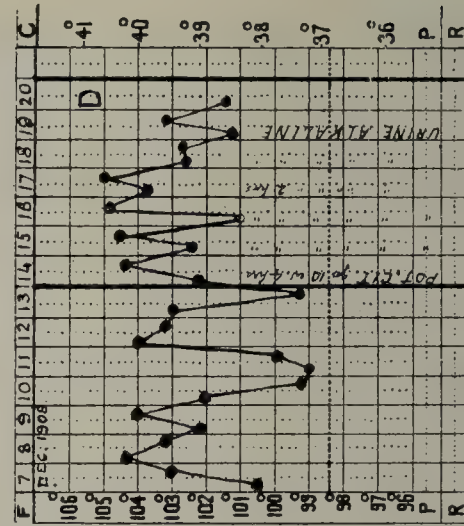


FIG. 136.—Acute pyelonephritis, showing absence of effect of alkaline treatment. (Boy of 7 months.)

pus may only be present in the last few drops evacuated from the bladder. This fact must always be borne in mind when passing the catheter so that the bladder is thoroughly emptied; otherwise no pus or organisms will be found and the condition missed. Should one side only be affected, the pus may from time to time disappear for a day or two, owing to the ureter of that side becoming blocked; in these circumstances, however, there is usually an exacerbation of the symptoms.

The *diagnosis* depends on two things. *First*, on the presence of pus and colon bacilli or other organism in the urine along with the severe general symptoms already described; and *secondly*, on the absence of any signs of organic disease outside the urinary tract which could account for the symptoms. In severe subacute diseases, such as tuberculous meningitis, pus and bacilli are often found in the urine from a terminal cystitis. On the other hand, acute pyelo-nephritis in young infants is often mistaken for meningitis, because of the vomiting, squinting, drowsiness, occasionally convulsions, and restlessness which are present. When large quantities of pus and colon bacilli are found in the urine during the early days of life, along with a palpable hypertrophied bladder, this signifies *Bacillus coli* infection of a congenital hydronephrosis—a condition which is always fatal.

In strong children over two years old, the *prognosis* is probably always good. In infants under one year, and especially when badly nourished, the prognosis is grave: they frequently succumb to some intercurrent infection, *e.g.*, broncho-pneumonia, and in them there is a marked tendency for the condition to proceed to abscess formation. When the disease is overlooked and left untreated, recovery certainly takes place in some cases, but the mortality is probably very much higher than in those that are treated. Relapses are common in untreated cases, and even also in those which are treated by antiseptics or alkalies.

Treatment.—In the first place, everything should be done to cause an abundant flow of urine and so flush the urinary tract. The child should have an abundance of fluid, and hence it is advisable to give milk freely diluted with barley-water. If the child refuses to drink water freely, water must be given by the stomach-tube or by rectum. The bowels should be attended to so that satisfactory daily evacuations are obtained.

Many forms of medicinal or specific treatment have been suggested:—

Alkalies.—At the present moment the most popular form is the administration of citrate of soda or potassium, or bicarbonate of soda, or a combination of the drugs, in doses sufficient to render the urine alkaline. Doses of 10 gr. four or six times daily may be commenced with and increased till the desired effect is obtained. At times as much as 240 gr. per day are necessary. Citrate of soda or potassium when given in large quantities are apt to induce diarrhoea, and hence when large amounts are required it is advisable to employ a certain proportion of bicarbonate of soda. There is a tendency for the urine to become acid again within three to seven days after it has begun to be alkaline, and thus a constant watch must be kept on the reaction of the urine. The possibility of alkalosis developing when large amounts of alkali are being administered must always be borne in mind.

It is generally held that it is the urine becoming alkaline which brings about a cure (Figs. 131 to 133). Although the fever may subside when the urine becomes alkaline, this is by no means invariably the case (Fig. 136). There is, however, no doubt that there is greater chance of recovery when the urine can be rendered alkaline than when this is not possible, as the following analysis of 73 cases treated with alkalies shows.

Mortality according to ability to alkalinise the Urine.

	Deaths.	
	No.	Per Cent.
In 30 cases urine rendered permanently alkaline	3	10·0
„ 53 cases urine rendered alkaline at some time	13	24·5
„ 20 cases urine never rendered alkaline . . .	11	55·0

It is doubtful, however, if the urine can be made sufficiently alkaline to hinder the growth of the organisms. It is more probable that the ease with which the urine can be rendered alkaline is merely a measure of the extent of the renal mischief. As noted above, the highest death-rate occurred in these cases in which the urine could not be made alkaline. If the disease is severe the activity of the kidney is impaired, and the excretion of the alkali as well as the diuretic effect of the citrates will be interfered with. It is possible, of course, that the alkaline reaction

causes the tissues to be bathed in a bland fluid, and in this way enables them to combat the infection more effectively.

Urinary Antiseptics.—(a) The *oral administration* of salol, urotropin, helmitol, hexamine, and hexyl-resorsinol has been credited with rendering the urine antiseptic through the liberation of formaldehyde. It must be remembered that with the exception of salol this only occurs in an acid medium, and hence no alkalies must be prescribed. If the urine is alkaline acid phosphate of soda should be administered. On the other hand, in the case of salol the urine must be rendered alkaline.

In our hands this method of treatment has been disappointing, and at times the urinary antiseptic seems rather to do harm from possible irritation as evidenced by hæmaturia. While of 63 cases treated with alkalies 46 per cent. recovered, of 26 treated with antiseptics only 23 per cent. recovered.

Some writers recommend that urinary antiseptics with acid phosphate of soda and alkalies should be given alternately, and sometimes this manœuvre would appear to be followed by benefit.

(b) The *intravenous injection* of internal antiseptics—proflavin and sulphato-violet—were both tried at the R.H.S.C., Glasgow. A cure was obtained with the latter preparation in 4 out of 9 cases treated. The dose administered varied from 2 to 4 c.c. of a 1 per cent. solution, and the number of injections between three and nine. In 5 cases there was no result, and in two of these the intravesical installation of protosil (p. 441) later was followed by a cure. With proflavin an undoubted antiseptic effect was obtained, but unfortunately it was only very temporary.

Blood Transfusion.—In view of the reputed value of this procedure in septicæmia it was tried at R.H.S.C., Glasgow, in twelve children. In no case, however, was there a cure, and in only one did there occur any improvement in the pyuria. Eight of the children died. It is possible that in some of the cases this measure was adopted too late, but this was not so in all of them.

Vaccines and Sera.—Serum treatment has been recommended by various writers. We have had no experience of its use. Vaccines which were so highly lauded by Gorter¹ have proved

¹ E. Gorter, 17th *Internat. Cong. Med. London*, 1913 (Sect. Dis. Child., Pt. II., p. 121).

disappointing in our hands. Occasionally they have seemed to help. But this beneficial effect was true especially of the acute cases which, it should be noted, may recover spontaneously.

Ketosis and Acidosis.—The most recent method of treating pyuria is by the induction of either ketosis or acidosis, and many writers record good results in otherwise incurable cases.¹ We ourselves, however, have had rather disappointing results from this line of treatment.

Ketosis is induced by means of a ketogenic diet (Appendix F, p. 1028), the effect of which is variously ascribed to the antibacterial action of the acetone bodies themselves and to the lowering of the *pH* of the urine. A ketosis induced in this manner usually passes off in a few days (p. 554), so that if it is to be effective it must be so during the first day or so after the commencement of the diet. For this reason it has been suggested that the acidotic effect should be increased and maintained by the administration of some acidosis-producing drug (chloride of ammonium—see pp. 550 and 558). With this drug, however, there is no resultant acetonuria, so that if it has any effect—and from the published records such would seem to be occasionally the case—acetone is not the essential factor. It is possible that diuresis, which is an accompaniment of ammonium chloride acidosis, may play some part in the result.

CYSTITIS.—This may be of a simple catarrhal variety, or it may be due to a growth of *Bacillus coli* or some other pyogenic organism, either pure or as a mixed infection.

Simple catarrhal cystitis, due, apparently, to concentrated or ammoniacal urine or to a chill, sometimes occurs in young children. It is rather commoner in boys than in girls, and it is occasionally important as a cause of persistent incontinence of urine. The symptoms may be extremely slight, the microscopical examination of the urine showing only mucus corpuscles, some epithelial cells, and a few leucocytes. The treatment consists in regulation of the diet, the administration of diluents, and the careful avoidance of cold.

Cystitis due to pyogenic organisms is a much commoner

¹ H. F. Helmholz and A. L. Clark, *Proc. Staff Meetings, Mayo Clinic*, 1931, vi., 605; H. Cabot, *Lancet*, 1932, i., 1308; H. F. Helmholz, *Journ. Amer. Med. Assoc.*, 1932, xcix., 1305; D. Band, D. M. Dunlop and I. L. Dick, *Proc. Roy. Soc. Med.*, 1932-33 (Sect. Urology, 1).

condition in the older child, at least as a primary affection, than suppurative nephritis, although it is not improbable that even in the infant it is the precursor of many examples of suppurative nephritis. The symptomatology and the reaction to treatment in very many cases admit of no other conclusion.

Just as in suppurative nephritis, the *Bacillus coli* is the usual etiological organism, although the streptococcus, staphylococcus, or *Bacillus proteus* may also be responsible. It is probable, too, that, just as in the case of suppurative nephritis, the infection in the primary cases is conveyed by the blood stream.

The child usually complains of frequency of micturition with dysuria, especially towards the end of the act, and perhaps some hypogastric pain. A few drops of pure blood may escape at the end of micturition. There is in uncomplicated cystitis no fever and an absence of toxic symptoms. Palpation over the bladder may elicit tenderness. The urine is dark in colour, due to the frequent presence of blood, and contains pus and organisms. When the infection is due to the *Bacillus coli* the reaction of the urine is acid, but if due to a mixed bacterial flora consequent on some urethral stricture with retention, the reaction may be alkaline.

Uncomplicated cystitis is most amenable to treatment. Extension upwards with the development of the so-called "surgical kidney" may, however, result, especially if proper treatment is not carried out. The same general treatment as was applicable for suppurative nephritis should be employed. Confinement to bed, an abundance of fluid, and a bland diet are essential, and the patient will be more comfortable if alkalies are administered. The most effective therapeutic measure in our hands has been the local application of protosil or neo-protosil. After thoroughly emptying the bladder by catheter 10 c.c. of a 10 per cent. solution of either of the above drugs are slowly introduced into the bladder and retained as long as possible. This procedure is repeated once daily for a week when, as a rule, all pus and organisms will be found to have disappeared. The efficacy of this method of treatment is demonstrated by the results obtained in twenty-six examples of cystitis. A cure was obtained in 88.4 per cent. of them.

If pus and organisms persist a second course of treatment should be applied, and if this fails a review of the case anew is

called for. A further search for tubercle bacilli may clear up the diagnosis, or radiology or cystoscopic examination with pyelography may reveal a hitherto undetected calculus in the bladder, some foreign body or some abnormality in the bladder or ureters interfering with proper drainage.

Glycosuria.—Sugar in the urine may often be found in cases of alimentary intoxication in infancy. When it occurs in any quantity in a child, without a distinct alimentary cause, its presence is of grave significance, as it generally indicates diabetes mellitus. Very rarely, a degree of glycosuria occurs as a family disease, and seems to have little or no bad effect on the general health. Sugar may be detected in the urine in a large proportion of cases of tuberculous meningitis, shortly before death (Still); also in some cases of tumours in the pancreas and pituitary gland.

Diabetes Mellitus is considered at p. 525.

The Kidneys.

NEPHRITIS.—The whole subject of nephritis at the present moment is in the melting-pot. Until it is possible to correlate the symptomatology and biochemical changes with the histological lesions, there can be no satisfactory classification of the types of nephritis. Most of the pathological pictures are end-results, whereas the chief interest for the clinician is to obtain a knowledge of the early stages, and to contrast the changes in those who recover with those in the patients who succumb. At the R.H.S.C., Glasgow, an attempt in this direction was made during a therapeutic test of the value of Edebohl's operation. At the time of the decapsulation pieces of renal tissue were removed and submitted for histological examination. In most instances there was a pan-nephritis or mixed nephritis (although one element might be more affected than another) as evidenced by both parenchymatous and interstitial changes, a view of nephritis which had been suggested by the investigation of the general post-mortem material.¹ It might be said that the behaviour and course of the disease would seem to be "determined more by the severity of the damage than by the particular kidney element involved."²

¹ K. J. Guthrie, *Glasg. Med. Journ.*, 1930, cxiv., 61.

² G. Campbell, *Arch. Dis. Child.*, 1930, v., 283.

It may be because of this tendency for all the kidney elements to suffer, *i.e.*, because nephritis in childhood is a pan-nephritis, that the various biochemical tests are of such limited value in prognosis. It is not possible to conclude from the urea or cholesterol contents of the blood, from the excretion of pigment intramuscularly injected (*pigment test*), or from the ability of the kidney to concentrate urea (*urea concentration test*) whether the case is parenchymatous in nature and thus likely to recover, or interstitial and tending to be progressive.

These various tests of *renal efficiency* are carried out in the child exactly as in the adult, and the normal values would seem to be little affected by age. Normal blood urea values vary between 20 and 50 mgrm. per cent. This shows a tendency to rise slightly after a meal, which may account for the higher readings. The normal blood cholesterol value in the child varies between 130 and 200 mgrm. per cent.; pigment excretion during the first two hours after the intramuscular injection of 6 mgrm. of phenolsulphonephthalein varies in the healthy child between 56 and 85 per cent. In the performance of the urea concentration test Calvert's modification, in which the variation in the urea excretion as a result of the administration of urea orally is noted rather than the obtaining merely of a concentration above 2 per cent., is to be commended. In health the urine shows a sharp rise in its urea content after the administration of urea, which is absent in disease. It is important also to correlate the urea output with the blood urea content, since, if the kidney is healthy, these two estimations will reveal a definite parallelism.

One of the most striking features of the various so-called renal efficiency tests is the inconsistency with which they react in the individual case. In one and the same child all the tests do not necessarily reveal either any, or the same degree of, renal dysfunction. Other disturbing characteristics in their interpretation are the wide variations met with in health, and the overlap of the values in the normal and the abnormal. This is explicable by the fact that at least two-thirds of renal tissue can be removed without causing any impairment of renal function. These various points are well illustrated in the comparative Table on p. 444, compiled by Dr Crawford from material studied at R.H.S.C., Glasgow.¹

¹ E. Crawford, *Glasg. Med. Journ.*, 1924, ci., 72.

Summary of Results of Different Tests in Health and in Acute and Chronic Nephritis.

	Pigment Test. Per cent. Excreted in Urine.			Urea Concentration Test. Urea in Urine per cent.			Blood Urea. Mgrms. per 100 c.c.		
	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.
Healthy children . . .	85	56	67.1	6.0	2.3	3.6	51.6	20.0	39.2
Reputed normal . . .	85	60	2.0	...	40	20	...
Acute nephritis . . .	94	21	51.0	3.6	1.2	2.07	357.0	22.0	73.0
Percentage of pathological findings in above .	70			44			70		
Chronic nephritis . . .	66	50	57	3.5	1.5	2.3	69.7	21.0	36.3
Percentage of pathological findings in above .	50			50			20		

One important feature in nephritis in childhood is the *metabolic change*, especially in so far as it affects the minerals calcium and phosphorus. The blood frequently shows a diminished calcium content and an increased proportion of inorganic phosphorus. These findings suggested a phosphorus retention, but, as Ford¹ has shown, there is a parallel and diminished retention of both of these elements which only slowly increases as recovery takes place. If, as is most likely, the same state of matters exists in chronic nephritis, a gradual and consequent impoverishment of bone-forming material must ensue, and it is this fact which probably explains the tendency to the development of rickets in chronic disease of the kidney (p. 282). A tendency to acidosis is not infrequent, especially if uræmia be present.

Uræmia is comparatively rare during childhood. It occurs in a larger proportion of chronic than acute cases, but is most frequently seen during the acute stage of the disease, and as a rule before any nephritic manifestation had been suspected and appropriate treatment adopted. Headache, vomiting, convulsions, coma, and amaurosis are the usual manifestations.

Cardio-vascular changes are also rare, or at least not striking during childhood. The systolic blood pressure may be increased and occasionally register 150 mm. of mercury (normal maximum 110 mm.) for a few days during the acute phase, but it is in

¹ F. J. Ford, *Arch. Dis. Child.*, 1931, vi., 209.

chronic mischief that this sign is most frequent, most marked, and most persistent. Cases with a systolic pressure of more than 120 mm. usually present hæmaturia. Cardiac hypertrophy is seldom met with unless in the case of chronic disease.

Optic neuritis is also rare and is usually associated with uræmic manifestations.

Clinically three types of nephritis may be met with: (a) *acute hæmorrhagic*, (b) *subacute non-hæmorrhagic*, and (c) *chronic interstitial*.

(a) **Glomerulo-Nephritis** (*Acute Hæmorrhagic Nephritis*).—Scarlet fever is usually considered the most important cause of this variety of the disease during childhood. Undoubtedly scarlet fever may cause a hæmorrhagic nephritis, and hence it is wise always to inquire as to recent sore throat with rash and fever, and to make a careful examination for traces of desquamation. But nephritis more often occurs quite apart from scarlet fever. In fact, in ordinary practice, scarlet fever is very seldom the etiological factor. On occasion nephritis may follow an attack of acute tonsillitis, but, since scarlet fever is seldom if ever transmitted by these patients, one cannot look upon the throat condition as scarlatinal in nature. Many children with nephritis have suffered from a previous and widespread impetigo. Quite often it appears as a primary disease brought on apparently by exposure to cold.

Sometimes nephritis is met with as a complication of whooping-cough, measles, diphtheria, erysipelas, chicken-pox, small-pox, mumps, septicæmia, glandular fever, and, especially during infancy, in association with congenital syphilis and gastro-enteritis. Considering the fact that acute nephritis and acute pneumonia are both frequently determined by chill, it is remarkable how seldom the two diseases occur together. Of 219 examples of acute Bright's disease and 416 of acute lobar pneumonia observed during a period of nine years, in only five of the children were both diseases coincident.¹

Symptoms.—The manifestation which most frequently attracts attention to this disease is œdema. This may appear quite suddenly, first in the face and later on in the limbs, without any discomfort having been complained of or anything abnormal having been observed in the urine. In other cases there is headache and vomiting with fever, perhaps pain in the

¹ L. Findlay, *Arch. Dis. Child.*, 1928, iii., 148.

back or abdomen, the latter in very young children having been mistaken for appendicitis and calling for immediate operation. On rare occasions uræmic convulsions or coma may be the first manifestation to draw attention to the condition. In other cases it is the passage of scanty dark-coloured urine which directs attention to the disease.

Course.—The prognosis in this type of nephritis (acute hæmorrhagic) is eminently favourable. In fact, it may be stated that the rate of recovery is directly related to the acuteness of the onset. Few of the cases pass into the subacute parenchymatous: this variety of the disease usually commences as such. Of a series of 25 consecutive cases of acute nephritis admitted during the course of a year to the R.H.S.C., Glasgow, 20 were dismissed completely recovered, 2 died, and 3 became subacute.¹ Greenlees traced 47 cases four years after their dismissal from the same hospital. Fourteen had died (five from uræmia); 35 were living, and in 26 examined the urine was free of albumin and the children in perfect health. Eighteen of these children had left hospital with the urine clear, but in 3 albumin had reappeared, whereas of 17 dismissed with albuminuria the urine was clear in 11.

The *treatment* of acute nephritis is no way peculiar in childhood. The patient should be put between blankets and kept at first on milk and barley-water. The skin must be made to act freely by the use of warm baths, hot packs, or hot-air baths. With the same object, liquor ammonii acetatis, or in bad cases pilocarpine ($\frac{1}{15}$ to $\frac{1}{4}$ gr. by mouth) may be given. The action of the kidneys is to be encouraged by alkaline diuretics and by giving fluids freely. The administration of large enemata of warm water is useful for this purpose. Poultices (plain or mustard) over the loins are also indicated, and dry cupping sometimes aids diuresis. The bowels should be kept freely open by saline purgatives and by occasional doses of calomel.

In some cases rapid and dangerous dilatation of the heart takes place. When this occurs, venesection with the removal of from 6 to 12 oz. of blood, according to the patient's age, is urgently called for and often saves his life.

In feeding cases of nephritis, it is important not to continue a slop diet for too long after the acute symptoms have subsided, and if all blood has disappeared and the urea concentration test

¹ G. Campbell, *Arch. Dis. Child.*, 1930, v., 283.

is satisfactory, even although there is still albuminuria, it is wise to increase the diet by giving fish, eggs, and white meat. Red meat and all meat extracts should be avoided.

(*b*) **Parenchymatous Nephritis.**—Cases of acute hæmorrhagic nephritis may pass into a chronic stage simulating parenchymatous nephritis and continue as such for months or years. Many cases, however, commence as parenchymatous nephritis, the chief characteristics of which are œdema and a severe degree of albuminuria without hæmaturia.

The onset is insidious, with general malaise, and nothing definite may be suspected till œdema appears and suggests examination of the urine, which is found to contain a large amount of albumin. Sometimes the œdema may definitely precede the albuminuria, a fact which supports the contention that œdema is due to some extrarenal factor. In this form of nephritis there is diminished excretion of chlorides, to which some authorities relate the œdema. Cardio-vascular changes are rare and uræmia seldom occurs.

The condition is exceedingly intractable and may vary much in intensity from time to time. After persisting for months or years septic peritonitis, pericarditis, or some other intercurrent disease may close the scene. In not a few instances, however, and for no apparent reason, the œdema and albuminuria disappear and the child makes a complete recovery.

In many of the cases the blood urea is normal or unduly low, the tests for renal efficiency reveal no impairment, and, when recovery takes place, it is suggested that the condition has not been inflammatory but of a degenerative nature. It is for this type of case that the term *nephrosis* has been introduced. Unfortunately, there is no pathological proof of any such condition. In our experience when an opportunity has presented itself of examining the kidney, as, *e.g.*, at the time of a therapeutic decapsulation (Edebohl's operation) there has always been evidence of pan-nephritis.

Treatment.—The exacerbations in chronic parenchymatous nephritis have to be treated like an ordinary acute attack. During the intervals, the action of the skin is to be encouraged by woollen clothing and regular warm baths. The child should, if possible, winter in a warm climate. It is unnecessary to endanger the general health by too strict a diet, provided articles which are rich in extractives and all spiced foods are

prohibited. When the œdema is troublesome, a salt-free diet may be tried (Appendix F, p. 1028), but this line of treatment is often most disappointing.

Decapsulation of the kidneys (Edebohl's operation) has been recommended in severe cases by Koplik and others. It is questionable, however, if the operation has any real value. In a series of 25 cases studied at R.H.S.C., Glasgow, the results obtained were practically the same as were observed in a similar series of non-operated cases.¹ The œdema very quickly disappears, but it would appear as if this were simply the result of the incision of the subcutaneous tissues which permits of its speedy escape.

(c) **Interstitial Nephritis** (*Cirrhotic Kidney*).—This is very uncommon in childhood. It may be secondary to acute nephritis but as a rule is primary either of congenital origin or commencing in early childhood. The patients have a history of having been delicate, dyspeptic, and stunted in growth all their lives, and are often the subjects of infantilism. In some cases rachitic changes appear (p. 282). In nephritis there is a defective retention of calcium and phosphorus with a resulting osteoporosis, so that rickets is predisposed to.

The main symptoms² are an increased amount of urine containing little albumin, occasional tube-casts and a low specific gravity, excessive thirst, no appetite, frequent headaches, and failure to gain weight. Œdema is rare, except with heart failure towards the end. Uræmia is the usual cause of death. In many cases, but not in all, the arterial tension is high and the heart hypertrophied. Blood urea values are often high (50 to 150 mgrm. per cent.) and renal efficiency tests occasionally reveal great impairment of function.

Tumours of the Kidney are commonest in early childhood and are rare after six years. Of eighteen examples observed at R.H.S.C., Glasgow, fifteen were blastocytomata, two were sarcomata, and one was a teratoma. The blastocytoma is a mixed tumour having the microscopic characters of both sarcoma and epithelioma, and is probably developed from a still undifferentiated embryonic tissue: occasionally formations like glomeruli are observed.

Generally few subjective symptoms, if any, are present until

¹ G. Campbell, *Arch. Dis. Child.*, 1930, v., 283.

² J. E. H. Sawyer, *Birmingham Med. Rev.*, 1903, N.S., II., 511, 549; C. H. Greene, *Amer. Journ. Dis. Child.*, 1922, xxiii., 183.

the later stages of the disease, although a considerable amount of blood may appear in the urine. A tumour of the adrenal displacing the kidney may closely simulate a true renal tumour. Sometimes an enlarged spleen suggests kidney, but this mistake should not be common.

When the condition is recognised early, excision may be advisable but the prognosis is very bad. Extremely few cases are on record in which the disease did not recur after removal.

Perinephric Abscess is not very common in childhood. It may be connected with disease of the spine or lower ribs; if on the right side, it is generally secondary to gangrenous inflammation of a high appendix, but it may arise spontaneously. In the early stages the urine may be clear and the symptoms are those of severe pain in the back simulating spinal mischief.

Renal Calculus is rare in young children. When present it is apt to escape recognition, owing to the difficulty of getting a clear account of the symptoms. If its presence is suspected, X-ray photographs should always be taken.

Hydronephrosis. — When an aseptic obstruction to the outflow of urine is complete it causes atrophy of the kidney only; when it is partial or intermittent it leads to hydronephrosis. Hydronephrosis may be congenital or acquired, and unilateral or bilateral according to the nature and situation of the obstruction.

Congenital hydronephrosis is usually bilateral and accompanied by dilatation of both ureters and dilatation and hypertrophy of the bladder. In the unilateral variety some congenital narrowing of, or pressure by an aberrant vessel on, the ureter will be found; in this variety the bladder is normal. In bilateral hydronephrosis, which almost invariably affects boys, the obstruction is found in the posterior urethra. Until little more than a decade ago it was generally stated that no organic obstruction could be found to account for the condition, and it was suggested that it was consequent on some functional disturbance of the vesical sphincter, *e.g.*, achalasia.¹ In 1919, however, Young and his collaborators² drew attention to the

¹ J. Thomson, *Brit. Med. Journ.*, 1902, ii., 678.

² H. H. Young, W. A. Frontz, and J. C. Baldwin, *Journ. of Urol.*, 1919, iii., 289.

presence in the posterior urethra of abnormal folds or valves of the mucous membrane which were probably of congenital origin (Fig. 137). Since then, many writers¹ have verified this observation, and it may be noted that in many of the examples

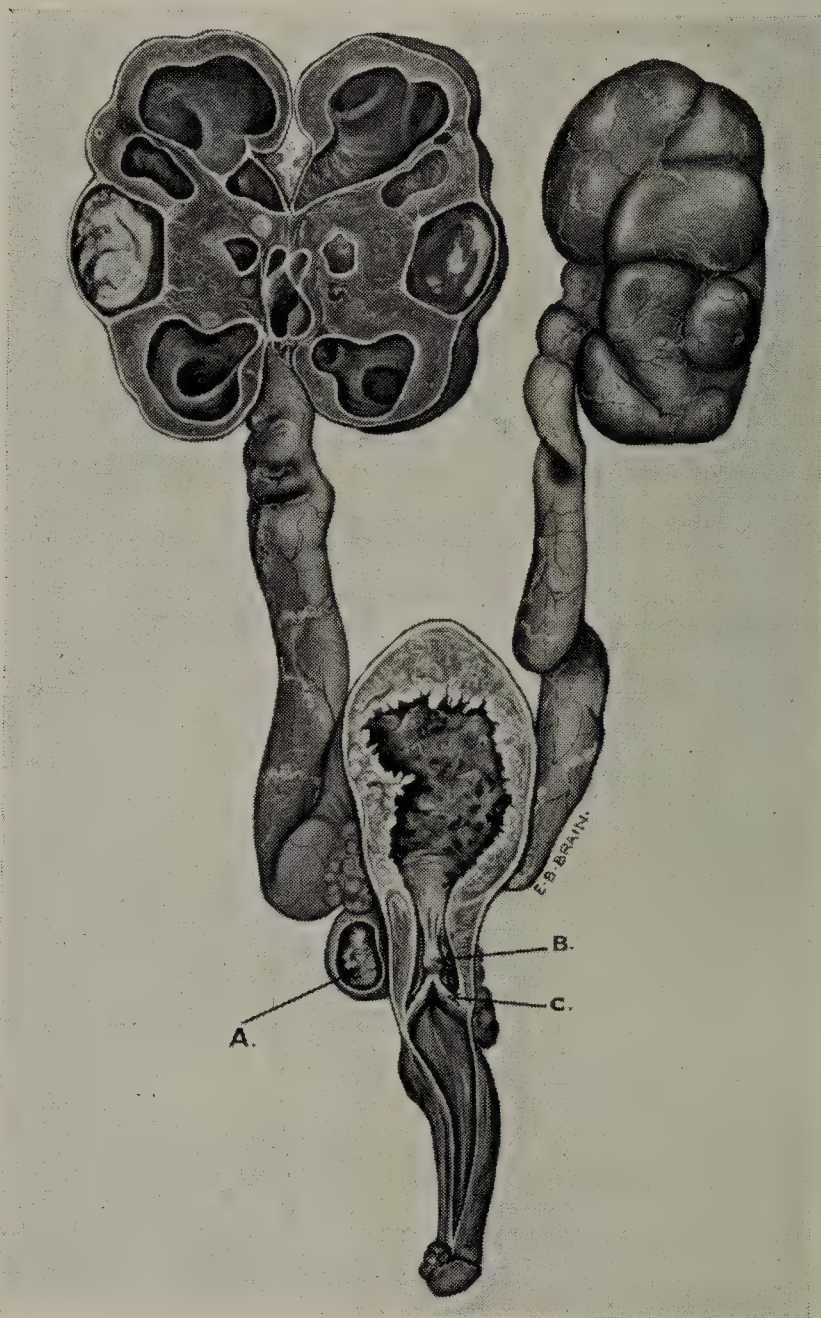


FIG. 137.—Baby G., aged 9 days ; showing
Congenital Urethral Valves.

- A. Perivesical abscess.
- B. Prominent verumontanum.
- C. Urethral valves.

(A. V. Neale, *Arch. Dis. Child.*, 1932, vii., 98.)

of idiopathic dilatation of the bladder and ureters, the urethra has not been examined.

¹ H. G. Bugbee, *Trans. Amer. Assoc. Gen. Urin. Surg.*, 1923, xvi., 235 ; F. J. Poynton and W. P. H. Sheldon, *Arch. Dis. Child.*, 1927, ii., 251 ; O. Addison, *Arch. Dis. Child.*, 1929, iv., 255 ; A. V. Neale, *Arch. Dis. Child.*, 1932, vii., 97.

Acquired hydronephrosis may also be unilateral or bilateral and accompanied or not by hypertrophy and dilatation of the bladder depending on the seat of the obstruction. If the obstruction (calculus, tumour of prostate) be situated in the urethra the bilateral variety ensues, with hypertrophy and dilatation of the bladder,¹ whereas if the obstruction be in one ureter (pressure of aberrant vessel or tumour or a calculus) then unilateral hydronephrosis results.

The patient may come under observation on account of dysuria. The age of onset of course depends on the particular cause. Addison² states that in the absence of phimosis, a pinhole meatus or some local inflammation or a calculus, a valvular obstruction of the urethra can be safely diagnosed in any case where the signs are those of chronic retention with overflow, with a history dating from infancy.

Only too frequently, however, the condition is disclosed through a pyogenic infection. This is, as we have already mentioned, very likely to happen if there is defective drainage (p. 434). When pyuria occurs in the new-born some congenital obstruction of the urinary tract is almost certain to be found. It has also been stated that such a cause should always be sought in any refractory example of pyuria.

Should a pyogenic infection not develop, the increasing hydronephrosis causes atrophy of the kidney tissue, which can be demonstrated by the application of the renal function tests. Defective growth, pallor, thirst, vomiting and finally acute uræmia are the usual symptoms.

The *treatment* depends on the discovery of the real cause, which necessitates X-ray examination, cystoscopy and pyelography. In the presence of pyuria the condition is hopeless, unless limited to one kidney, when excision is possible. If, however, the bilateral variety is detected early before there has been much destruction of renal tissue, the urethral obstruction may be relieved by operation with permanent benefit.

Intermittent hydronephrosis.—There is a class of case in which the tumour formed by the hydronephrosis suddenly disappears from time to time, along with a profuse discharge of urine of low sp. gr., and reappears later, after a varying number of weeks or months—its return being accompanied by vomiting and renal colic. The etiology of these cases is very

¹ A. V. Neale, *Arch. Dis. Child.*, 1932, vii., 97.

² *Loc. cit.*

obscure, but the therapeutic indications are generally fairly clear. If the recurrent attacks of pain and renal enlargement are becoming more frequent and more severe, and the child's health is suffering much from them, the kidney may have to be excised. If, however, as has often happened by the time the case is seen, the attacks are becoming fewer and less painful, and the child is fairly well, it is generally advisable to defer the operation indefinitely. For it is certain that the kidney is steadily undergoing atrophy and that in time its functions, and with them the attacks, will cease altogether.

Renal Tuberculosis.—Small tubercles are often found, post-mortem, in the kidneys in young children as part of a general tuberculosis. Much less frequently renal tuberculosis occurs as a more or less isolated affection in an otherwise fairly healthy child. It is most important to recognise these cases early, because the infection in them begins in one or other kidney and spreads from it to other parts, and treatment in an early stage may prevent this extension. Incontinence of urine is an early symptom, and later, frequent micturition and dysuria are very characteristic. Towards the end, pain may be felt in one or other loin. The urine usually contains an increase of cells—polymorphonuclear leucocytes or lymphocytes—and pyogenic organisms (*Bacillus coli*) in addition to tubercle bacilli. Hence in any example of pyuria of any duration and not reacting to treatment a search for the tubercle bacillus should be made. A twenty-four hour specimen should be obtained and the centrifugalised deposit examined for tubercle bacilli. If none are found, injection of the matter into a guinea-pig may give positive information. Pirquet's cutaneous or Mantaux's percutaneous reaction is of course helpful in young children. A cystoscopic examination with catheterisation of the ureters permits not only of the localisation but also of observing the extent of the disease.

Treatment.—In cases diagnosed as instances of early renal tuberculosis, great and permanent benefit is sometimes obtained from a course of old tuberculin injections; and this treatment is well worth trying if there are reasons against an operation. When the disease, however, is definitely one-sided, nephrectomy should always be performed without delay. Children stand the operation well, and the result is often entirely successful.

Renal Colic.—Distinct renal colic with the passage of uric acid crystals occurs occasionally in infants.¹ It is often accompanied by a considerable rise in temperature (Fig. 127, p. 417), obviously severe pain and general tenderness, sometimes with convulsions and with special pain on pressure over the region of the affected kidney, and retraction of the testicle on the same side. It rapidly recovers under alkaline treatment with diluents.

The Bladder.

In young children the bladder is situated so high that it is an abdominal as well as a pelvic organ. Special care must *always*, therefore, be taken to see that it is empty before tapping the abdomen.

Micturition.—As the kidneys are acting freely during later intra-uterine life, the infant often passes water immediately after birth. Not infrequently, however, the bladder has been emptied before or during labour, and no urine may be passed for the first twenty-four hours, or even longer. During the early months of infancy incontinence is, of course, the normal condition.

In the first two years the child passes water very frequently while awake, though during sleep he can retain it for two to six hours. By the third year it may be held during sleep for eight or nine hours, and, when awake, for two or three hours. The intervals between micturition steadily increase as the child grows older. Some infants have acquired control over their bladder by the time they are eighteen months or even a year old. In others this comes much later, but a great deal depends on the training the children receive. If a child cannot control his bladder to a considerable extent, however, during his waking hours, by his third year, he may be regarded as suffering from incontinence.

Retention of Urine.—Retention is not a very common symptom in childhood. In boys it may be caused mechanically by a calculus which has become impacted near the meatus; sometimes it is due to phimosis or to a congenital valvular obstruction of urethra (p. 451). It may also arise reflexly from irritation of the rectum by thread-worms, or from an anal fissure.

¹ R. A. Gibbons, "Renal Colic in Infants," *Med. Chir. Soc. Trans. London*, 1896, lxxix., 40.

Sometimes it is met with in enteric fever, and often during tuberculous meningitis. Occasionally it occurs, even in children as young as six years, as a hysterical symptom.

Suppression of urine is a common symptom in acute nephritis. It may, however, also occur in children for long periods without any serious disease of the kidneys—for example, when there has been much loss of fluid from diarrhœa, vomiting, or diaphoresis, and also sometimes after abdominal operations. If long continued, it may be treated by large enemata of warm water, alkaline medicines, and dry cupping.

Increased Frequency of Micturition (*Pollakiuria*).—A tendency to pass urine at too short intervals is often present along with enuresis, but it may occur alone, merely as a nervous symptom without any local cause. Sometimes it is aggravated by acidity of the urine, by the presence of uric acid or oxalate crystals, or by bacteriuria. Its onset in a little child should suggest examination of the urine for signs of cystitis from *Bacillus coli* infection, for it is not uncommon to find that frequent micturition has existed for a week or two before the onset of acute pyelitis; and prompt treatment will prevent the upward extension of the disease.

Pollakiuria may occasionally be relieved by belladonna and bromide, or, if the urine is acid, by its alkalinisation. In the majority of cases, however, treatment of the child's mental condition is the most important part of the treatment. The habit is generally associated with enuresis; and the fear of wetting himself keeps the child in a constant state of mental unrest, which greatly interferes with his recovery. If the child's clothing, the climate, and the surroundings generally were such that the habit could be entirely ignored and treated as of no consequence, it would probably cease rapidly in most instances. These children should have their pelvis, thighs, and legs kept thoroughly warm.

Incontinence of Urine. — *Enuresis* is a common and important symptom in early life. It is met with in a severe form in many types of mental defect and in organic nervous disease. Rarely it is a symptom of nocturnal epilepsy, diabetes, or tuberculous or other infection of the urinary tract. When it has existed from birth, some local malformation should always be looked for.

In the great majority of cases, incontinence of urine is a

pure neurosis, which, though possibly favoured by the presence of local irritation or slight derangement of the urinary tract or of the urine, depends mainly on an ill-regulated and badly-trained state of the child's nervous system, with a consequent mental flabbiness and lack of self-control. It is especially common among the upper classes, and the tendency to it is often hereditary. It is important to realise to what a large extent it is encouraged, in many instances, by the unconcealed apprehensions of the parents and by their injudicious suggestions, which increase the child's shame and distress, and diminish the self-confidence on which his recovery so largely depends. In young children the habit is seldom made so much of by their elders, and consequently is rarely so severe. Similarly, we often find that children who suffer badly from it at home, recover rapidly when admitted into a hospital ward, where their wetting is treated in a less emotional way.

The local abnormalities which may favour or aggravate the habit are numerous. There may, for example, be concentrated ammoniacal or highly acid urine, or bacilluria. A long redundant or adherent prepuce which is constantly the seat of inflammatory irritation, and narrowing or excoriation of the meatus, are other not uncommon auxiliary causes, even when no phimosis exists. Unnecessary circumcision is a not infrequent cause. Rectal irritation from thread-worms is sometimes to blame.

Chills from any cause are certainly important as predisposing causes, perhaps owing to their favouring the occurrence of slight vesical catarrh (Eustace Smith); and there is generally a tendency for the habit to be worse during the cold and wet seasons. Any influences which tend to cause restless sleep—such as emotional excitement before going to rest, too warm coverings in bed, or the presence of adenoid growths, may also have a predisposing influence. Recently it has been suggested that enuresis is at times an allergic manifestation.¹

In many cases the enuresis occurs to some extent during the day as well as at night, and, less commonly, it may be altogether diurnal. Generally, however, the micturition is normal, or merely too frequent, during the daytime, and the incontinence is nocturnal only. In a few instances there is a history of continuous enuresis from early infancy; but usually the child's

¹ G. W. Bray, *Arch. Dis. Child.*, 1931, vi., 251.

control has been normal for months or years before the incontinence returned.

Treatment.—In treating a case of enuresis we must always bear in mind the necessity of taking the patient's mental condition into account. One has known a bright, vigorous little child, who had been spoiled at home, whose enuresis was rapidly improved by the happy discipline of a good kindergarten. In a very few cases in older children, who are lazy and apathetic, the fear of punishment may do good. In the vast majority of instances, however, punishment does nothing but harm, and only aggravates the symptoms. Serious talks from his parents on the disgracefulness of the habit almost always make matters worse by concentrating the child's attention on the habit and making him exaggerate its seriousness. If an older child, who is indifferent about the matter, can be stimulated by hope of reward or otherwise to desire greatly to recover, and especially if he is made to feel that he is likely to do so, this has always a good effect. But when, as often happens, the patient is already painfully anxious to be cured, he must receive every possible encouragement, and be impressed with the fact that the condition is not so very terrible after all, and that it always gets right in time. Especially, he must be assured that there is no blame whatever attaching to him in the matter.

Although the mental management of the case is generally the most important part of the treatment, other measures are by no means to be neglected. They are often of the greatest benefit. In most cases this is largely because they encourage and help the patient to cure himself.

In beginning the treatment, obvious local abnormalities and general weakness must, of course, be dealt with first, and the urine examined. If cystitis is present, or the urine is abnormally acid or alkaline, or there is glycosuria, these conditions should be treated. A narrow meatus requires dilatation, and an excoriation careful dressing with a suitable ointment. Phimosis and irritation from the retention of smegma are to be treated by separation of adhesions, or, if necessary, by circumcision.

The influence of chills in prolonging the habit is not to be forgotten. A cold douche carefully managed (p. 989) is sometimes very useful in bracing up the system; but, if it is carelessly given, it may be a source of renewed chills.

The child should always be warmly clad, especially about the legs and pelvis. When the enuresis is nocturnal only, it is well to encourage the patient to practise holding his water for as long as possible during the day.

In most cases, the amount of fluid in the diet should be limited, especially during the latter half of the day; and the child should not be allowed to drink anything after 5 or 6 P.M. He should also be roused to pass water about 11 or 12 at night.

When, as very often happens, no obvious indication for special treatment exists, we must try the effect of tonics and of drugs which act on the nervous mechanism of the bladder; in many cases these are very helpful.

Preparations of belladonna or atropine should generally be first tried, especially in older children; and to be of use in nocturnal enuresis they must be given in full doses, so that some dilatation of the pupil and dryness of the throat are present at bedtime. From 10 to 15 minims of the tincture of belladonna may be given at night to a child of five or six years; and an older child may have from 15 to 20 minims in the afternoon, and again at bedtime. If this is not enough, the dose may be gradually increased. A solution of atropine is sometimes preferred as being less variable in strength than the tincture. Holt recommends a solution of 1 gr. in 2 oz. of water. He begins with 5 minims of this in the afternoon and evening, for a child of five years, and gradually increases the dose to 10 minims. The medicine should be continued for some time after the enuresis ceases, and then be slowly diminished in amount. The addition of bromide is often useful.

In cases in which belladonna or atropine fails, we may, in older children, try cantharides, in the form of a blister over the sacrum, or give 1 or 2 minims of the tincture thrice daily. Liquid extract of ergot (20 to 30 minims, thrice daily) is sometimes surprisingly effectual in children between three and five years in whom belladonna has failed; although often it has no effect. Liquor strychninæ, also, in 2 to 5 minim doses, may frequently be given with advantage along with belladonna. Liquid extract of *rhus aromatica* (10 to 30 minims, thrice daily) is also worth trying; it often aggravates the symptoms at first, and requires to be persevered with for many weeks.

The injection of normal saline into the cauda equina, with

the idea of stimulating and improving the tone of the vesical sphincter, has been recommended. Like all methods of treatment this variety is not always rewarded by success. One of us (L. F.) obtained by this method of therapy occasionally remarkable results, but equal success followed the subcutaneous injection of saline into the mons veneris. It appeared, therefore, as if it were the mere fact of an operation being performed which exerted a psychic effect.

The treatment of enuresis by thyroid was first suggested by Hertoghe,¹ and has been recommended also by L. Williams² and others. Our experience agrees with that of Firth,³ that it is specially useful in mentally defective children who have suffered from the habit all their lives. The dose should be small at first, beginning with $\frac{1}{4}$ gr. or $\frac{1}{2}$ gr., once a day. It may be gradually increased, at fortnightly intervals, up to 2 gr. twice daily. The larger doses are apt to increase the enuresis in some cases. The frequency and obstinacy of incontinence of urine in young cretins under thyroid treatment are interesting in this connection (p. 512).

Bray⁴ claims that cases not reacting to curtailment of the fluid intake and the administration of belladonna will usually be found to be due to hypersensitiveness to some particular article (food, inhalations, etc.), and that by desensitisation to the special substance a cure is obtained.

Dysuria.—The upper urinary passages are very tender in little children, and concentrated urine or uric acid crystals may give rise to much obscure local pain and general distress. This is to be watched for in cases of frequent vomiting with pyrexia, especially in babies (p. 416). It can often be rapidly relieved by diluents and alkalies. In boys, pain on passing water is often due to phimosis or local irritation, and disappears when these conditions are attended to. It is, however, the characteristic feature of congenital valve-like urethral stricture (p. 451). Spasmodic dysuria arises, not very rarely, from rectal irritation. It occasionally occurs also as the first symptom in tuberculous meningitis. Painful and frequent micturition may,

¹ Hertoghe, *Bull. de l'Acad. Roy. de Méd. de Belgique*, 27th April 1907, 267.

² L. Williams, *Lancet*, 1909, i., 1245.

³ D. Firth, *Lancet*, 1911, ii., 1619.

⁴ G. W. Bray, *Arch. Dis. Child.*, 1931, vi., 251.

of course, be due to cystitis, to vesical calculus, and in older children to renal tuberculosis. They are also common and important symptoms in appendicitis and pelvic peritonitis.

Severe *spasmodic dysuria* is sometimes met with in little girls. The child has an urgent desire to pass water, and screams with pain when she tries to do so. This ailment can usually be speedily relieved by administering hyoscyamus and potash, or an opiate, and by giving diluent drinks and a hot hip-bath.

CHAPTER XIX

DISEASES AND DEFECTS OF THE GENITAL ORGANS

Male

Adhesion of the Prepuce to the Glans Penis.—In the normal new-born child the prepuce is adherent to the glans. When the adhesion extends to near the meatus, the opening of the prepuce looks smaller than it really is, and the condition is apt to be mistaken for phimosis and to lead to unnecessary operation.

In such cases, when the adherent surfaces are separated with a probe, the foreskin retracts quite freely. If there is no local irritation the adhesions may safely be left alone, as they will do no harm and always disappear in time. If, however, they are giving rise to much accumulation of smegma, and especially if balanitis occurs, the surfaces should be gently separated, and the mother instructed to draw back the prepuce daily for a few weeks and apply a disinfectant, *e.g.*, carbolic oil or ointment. If, after separation of the adherent surfaces, satisfactory retraction of the prepuce is still impossible, the condition is one of phimosis and calls for further treatment.

Phimosis.—Abnormal narrowing of the opening of the prepuce occurs in varying degrees. When it is extreme, it hinders the free passage of the urine and may lead to balanitis and dysuria. Phimosis is credited with being the exciting factor in a wide variety of conditions: retention of urine, enuresis, insomnia, masturbation, convulsions, pyloric stenosis, etc., etc. This is exceedingly doubtful and it may be truly said that it is diagnosed often when non-existent and the operation of circumcision only too frequently unnecessarily carried out.

Treatment.—Dilatation of the prepuce has been strongly recommended by some, and it sometimes succeeds in the milder cases; but it is a troublesome proceeding and often fails. Early circumcision is generally to be preferred.

Circumcision, when necessary, should be done early—preferably within the first few months of life. The operation need not be described here, but it may be mentioned that enough of the prepuce should invariably be left to cover the corona. The corona glandis is a delicate structure, and it is certainly a mistake to remove unnecessarily the admirably efficient provision which Nature has made to guard it from friction and injury. From exposure of the glans ulceration commonly results with, on cicatrisation, narrowing of the meatus requiring further treatment.

While, in suitable cases, circumcision is a necessary and harmless operation, it is difficult to sympathise with those who recommend that it should be performed indiscriminately on all male infants. A great deal too much has often been claimed for it. In beginning the treatment, in cases of obstinate masturbation, circumcision may prove a useful adjunct to other measures as, by the pain it causes, it ensures a complete cessation of the bad habit for a short time. It has, however, no permanent, or even long-continued effect on the habit; and in some cases it even favours its beginning and continuance. Circumcision is also disappointing in its effects as a treatment of enuresis. It certainly stops it in some cases, but in many cases the incontinence is aggravated by the operation; and, in not a few instances, we have seen enuresis begin for the first time shortly after circumcision had been performed for other reasons.

Balanitis is often predisposed to by a neglected phimosis. It may be accompanied by considerable purulent discharge and some pain on passing water. Generally, the inflammation subsides rapidly when the affected cavity is frequently syringed with normal saline solution or boracic lotion. If the balanitis goes on recurring, circumcision is indicated; but it should not be done while the parts are inflamed. An *ulcer on the glans penis* round the entrance to the meatus is a common result of the irritation of ammoniacal urine on the diapers in little boys, especially in those who have been circumcised (p. 427). It is often slow in healing, and occasionally extends into the meatus and leads to a cicatricial contraction there. It may also be a cause of incontinence. Ulcers of this kind should be dressed with boric ointment and protected from friction and wet diapers. If, as often happens, the ulcers refuse to

heal with this treatment, the meatus should be slit up, and stitches put in to prevent contraction on healing.

A Congenital Narrowing of the Meatus is an occasional cause of enuresis. It requires gradual dilatation.

Urethritis may be due to gonococcus infection, or to extension of the inflammation into the urethra in cases of simple balanitis. It rarely occurs in a painful form in cases of bacillus coli infection of the urinary tract.

We have each seen one case of what seemed to be **Prostatitis** with abscess in boys of nine and ten years which recovered completely. Generally, in the rare cases in which children have prostatic enlargement, it is caused by malignant disease or tuberculosis.¹

Other Malformations, such as hypospadias, extroversion of the bladder and milder degrees of epispadias, are mainly of surgical interest. Extroversion of the bladder is best treated by transplantation of the ureters into the rectum. The result of this operation is often satisfactory in spite of the risk of subsequent bacillus coli infection of the urinary tract.

Delayed Descent of the Testicles.—Normally the testicles pass down the inguinal canal during the ninth month of intra-uterine life. Not uncommonly, however, the process is delayed, so that one or both sides of the scrotum are empty at birth. When this is so, the testicle sometimes descends during the first few months without treatment; and this may occur any time up to puberty or even later. If it is delayed beyond the first year, the testicle is generally accompanied by a hernia. Sometimes the organ becomes arrested in the inguinal canal, and in rare instances it goes astray and is found in the groin or perineum. One or both of the testicles may remain permanently in the abdomen. Such retained testicles are generally, though not always, functionless.

The recognition of non-descent of the testicle is not always easy on the first examination, as the organ may be temporarily retracted into the inguinal canal. A careful examination after application of warmth to the part will usually, however, make the diagnosis clear.

Treatment.—When the descent of the testicle is incomplete, it may sometimes be assisted by repeated careful attempts, made several times a day, to bring it down by manipulation;

¹ G. H. Edington, *Brit. Med. Journ.*, 1909, ii., 754.

attempts to replace the testis should always be made before the age of spermatogenesis. The treatment, however, is usually a matter for the surgeon. As the testicle in these cases is frequently functionless, it is often well to have it removed. It would appear that a retained testis is more liable to malignant disease. In deciding about its removal the question of the effect of its internal secretion has to be borne in mind.

Hydroceles of various kinds are common, especially in young infants, and they rarely cause much trouble. Hydrocele is almost constant in the new-born, but it usually disappears spontaneously. If the condition persists surgical treatment may be necessary.

Acute Orchitis.—The common form of orchitis due to mumps is practically unknown in children under twelve, and rare under fourteen. It sets in about the end of the first week, at the time when the parotid swelling is disappearing, and it is often accompanied by severe general disturbance with high temperature and much local pain. It frequently results in atrophy of the affected testicle. The treatment consists in hot fomentations, with support of the testicles by pads of cotton wool; and sometimes by incision of the fibrous capsule, to relieve tension.

Chronic Orchitis is not very uncommon in babies with congenital syphilis. It is generally bilateral and is often complicated by epididymitis and hydrocele. The testicle is enlarged, hard, and painless. The condition usually seems to recover completely under specific treatment.

Tuberculous affections of the testicle and epididymis occur occasionally, especially in infants. They may appear as part of a generalised infection, or, usually in older children, the disease may be limited to the genito-urinary tract. Generally both testicles are affected. When suppuration occurs, as often happens, the diseased parts should be excised. In cases of tuberculosis of the testicle, the prostate should always be examined, as it is frequently also affected.

Tumours of the Testicle may be congenital or acquired. The former may be dermoid cysts, myomata, or lymphosarcomata. Non-congenital tumours of the testicle occur mostly in older children. They are usually round-celled sarcomata of a very malignant character, which require excision as early as possible and are very apt to recur.

Female.

The Mammæ.—All new-born babies have a secretion of milk in their breasts and, curiously, it is more marked in boys than in girls. The fluid secreted resembles diluted woman's milk in composition, and, under the microscope, looks like colostrum. The secretion begins in intra-uterine life, but swelling of the breasts is usually only noticed some days after birth. This increases until about the ninth day, then slowly diminishes, and is usually gone about twenty days after birth. Occasionally it lasts much longer; one of us (J. T.) has seen greatly enlarged and secreting mammæ in a child of four months.

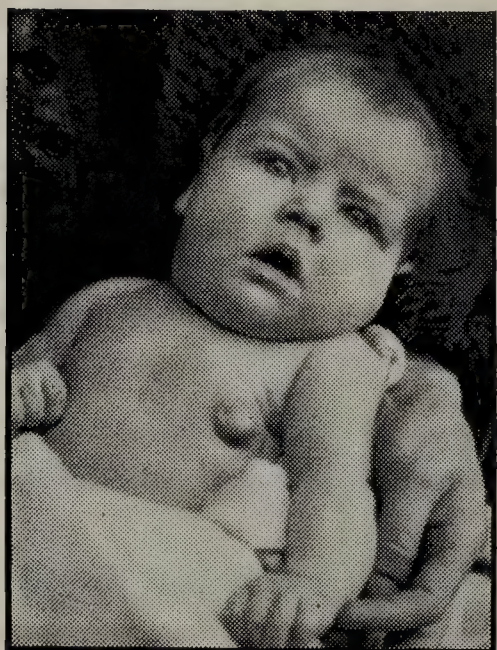


FIG. 138.—Mammary Abscess.
(Baby of 6 weeks.)

The swollen breasts should be carefully protected against injury, and kept surgically clean. Occasionally an abscess forms in one or both of them through the entrance of pyogenic organisms (Fig. 138). These abscesses generally heal rapidly when opened. If the child is sickly and ill-cared for, however, serious sloughing may occur.

Girls between nine and twelve years old occasionally suffer from a condition known as *irritable breast*. One of the mammæ enlarges and becomes tender for a time, and after an interval the other may be similarly affected. The condition is harmless and requires no special treatment.

Congenital Absence of the Breast is found on the affected side in most cases of "unilateral pectoral defect" (p. 847); and, when this condition is present, the pectoral muscles and costal cartilages should always be examined. Generally the nipple remains in a rudimentary form, but, in rare instances, no trace of it is present (Figs. 252 and 253, pp. 847 and 848).

Supernumerary Mammæ, either on one side or on both sides, are not infrequently observed.

Adhesions of the Prepuce of the Clitoris usually give rise to no symptoms, though some writers have attributed many reflex nervous disturbances to them.

Not very rarely, the opening of the vagina is closed in young infants or older girls by a simple epithelial **adhesion of the labia minora**. This condition can be put right at once by gently separating the parts with a probe.

Congenital Prolapse of the Uterus is a rare condition, which occasionally occurs as a complication of spina bifida in the lumbo-sacral region along with rectal ectropion and double club-foot. The children always die within a few weeks.¹

Vaginal Hæmorrhage is the least serious of the manifestations of the hæmorrhagic disease of new-born children (Chapter XX, p. 492). Usually it sets in within the first six days and rarely after the twenty-first. It only lasts for a few days and is never serious. It is probably connected with the congestion of the pelvic veins, which results from the cessation of the circulation in the umbilical arteries on ligature of the cord. A similar discharge of blood from the rectum may accompany it. It generally requires no special treatment.

Precocious Menstruation may occur at any age from eighteen months onwards, and the discharge may recur at monthly intervals. It may be accompanied by other indications of premature sexual development, and is sometimes a symptom of cortical hypernephroma (p. 522).

Simple Catarrh of the Vulva and Vagina is not uncommon. It may arise from dirt, friction, or thread-worms, or from the contact of irritating urine or fæces, and it is especially apt to occur after such debilitating diseases as measles. It usually recovers after a week or two of douching with boric lotion and the application of a mild mercurial ointment.

Gonorrhœal Vulvo-vaginitis is an exceedingly troublesome and obstinate complaint which may be met with at any age. It is in most cases acquired accidentally from contact with infected bed-clothes, sponges, or thermometers.

In America it has been found so extremely contagious that, when a case is admitted into a ward, others always occur within a few days in spite of the most careful precautions.² In this country the tendency to spread in this way seems not to be quite so great, but it is still considerable.

¹ J. W. Ballantyne and J. Thomson, *Amer. Journ. Obstet. and Dis. Wom. and Child.*, 1897, xxxv., 2.

² L. Emmet Holt, *New York Med. Journ.*, 18th and 25th Mar. 1905, 521 and 589.

In some instances the discharge is profuse, in others it is scanty. Young babies usually show no sign of local discomfort, but, in older children, dysuria is often complained of. The distribution of the disease varies in different cases; sometimes the cervix is much affected and the vagina comparatively free. It is rare for the disease to spread to the tubes or to the pelvic peritoneum. The main complications are conjunctivitis, arthritis, and teno-synovitis (p. 85); endocarditis and peritonitis are less frequent.

Treatment.—The child should be kept in bed, the general health attended to, and local remedies applied. The treatment must be most carefully and assiduously carried out, as the disease is very intractable. Good results have been reported from the use of a detoxicated vaccine, but, generally, the measures found most efficacious consist in douching with weak antiseptic lotions, swabbing with stronger solutions of antiseptics, and occasionally the insertion of pessaries.

Copious douching of the vagina and superficial parts with such mild antiseptic lotions as a 1-per cent. solution of protargol, 1 in 10,000 hydrarg. perchlor., saturated solution of boric acid, and 1 in 300 lactic acid lotion, is certainly beneficial. Probably, however, the good result depends fully more on the mechanical action of the lotion in cleansing the parts than on any antiseptic effect on the gonococci. The douching should be repeated four times a day. Another useful measure is the filling of the vagina with pure eucalyptus oil and keeping it in for a minute or two by holding up the child's legs and pelvis.

If, on examination of the deeper parts, localised areas of severe inflammation are discovered, these should be swabbed with a 10-per cent. solution of protargol, or a 1-per cent. lotion of lactic acid, on a dressed probe. It is generally advisable to alternate the use of different antiseptics from time to time. In convalescent cases, lactic acid pessaries are sometimes helpful. A weak mercurial ointment should be applied to the external parts.

When the treatment is thoroughly carried out, recovery may take place in from three to six weeks; but often, in spite of great care and trouble, the disease lasts for months.

CHAPTER XX

THE BLOOD AND ITS DISEASES¹

The Blood in Infancy and Childhood

DURING the first four years or so of life the child's blood differs from that of the adult in various ways.

At birth the blood is more concentrated, the hæmocytes averaging 5,500,000, and the total leucocytes 20,000 in the cubic millimetre, while the hæmoglobin is about 110 per cent. At this stage the red corpuscles show a tendency to inequality in size, and during the first few days some nucleated red cells are normally present. Among the leucocytes, polynuclear forms predominate, and the lymphocytes are relatively few in number.

This state of things does not last long (Fig. 139). Within the first fortnight the number of the hæmocytes has fallen to 5,000,000 in the cubic millimetre, at which figure it remains through life. The total number of the leucocytes diminishes until it reaches 10,000 per cubic millimetre at the end of the first week, and then rises again steadily to 15,000 by the end of the sixth month.

From this age till the end of the fourth year, any leucocyte count between 10,000 and 15,000 per cubic millimetre may be regarded as not abnormal. The average adult number (8000) is reached about the time of the second dentition. The proportion of the different forms of leucocyte also changes. During the first week of life the polymorphonuclears rapidly diminish and the lymphocytes gradually increase in number (Fig. 140).

¹ J. S. Fowler, "On the Diagnosis and Prognosis of some Forms of Blood Disease in Infancy," *Internat. Clinics*, 1901, 135, and "On the Splenic Anæmia of Infancy," *Brit. Med. Journ.*, 6th Sept. 1902, ii., 694; Robert Hutchison's Goulstonian Lectures, *Lancet*, 7th, 14th, and 21st March 1904, i., 1253, 1323, and 1402; J. H. Thursfield, "On Blood Diseases in Children," *Brit. Med. Journ.*, 26th Nov. 1921, ii., 873; Poynton, Thursfield, and Paterson, *Brit. Journ. Child. Dis.*, 1922, xix., 57, 128, and 178; O. Naegeli, *Blutkrankheiten und Blutdiagnostik*, Berlin, 1931, 5th edition; W. P. Lucas and A. H. Washburn, "Blood and Blood-building Organs," *Clinical Pediatrics*, vol. xi., New York, 1928.

The proportion of the hæmoglobin falls steadily from 110 to 120 per cent. at birth until it is only about 70 per cent. at the end of the sixth month, at which level it remains until eighteen

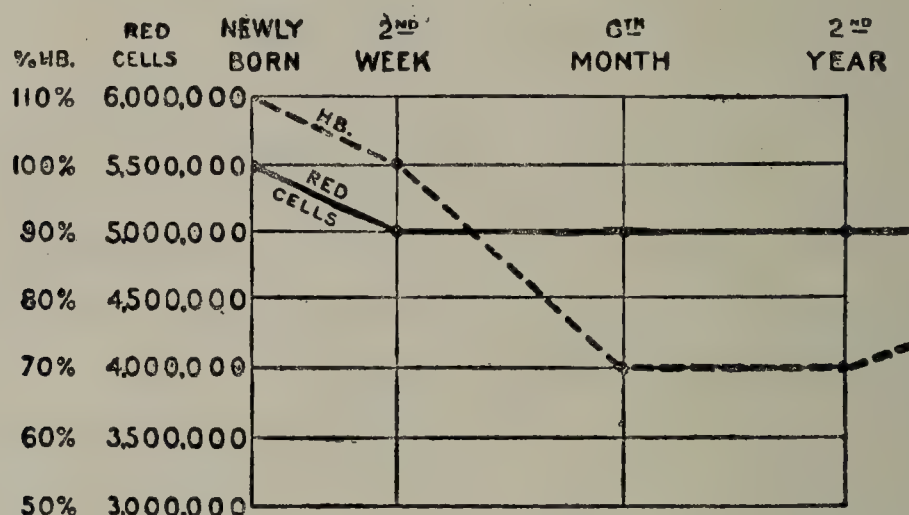


FIG. 139.—Proportion of Hæmoglobin and Red Corpuscles throughout infancy. (R. Hutchison.)

months, and thereafter slowly rises and gains the level of 80 to 85 per cent. about the sixth year.

We see from the above that the main normal characteristics of the blood in early childhood are the high absolute number of

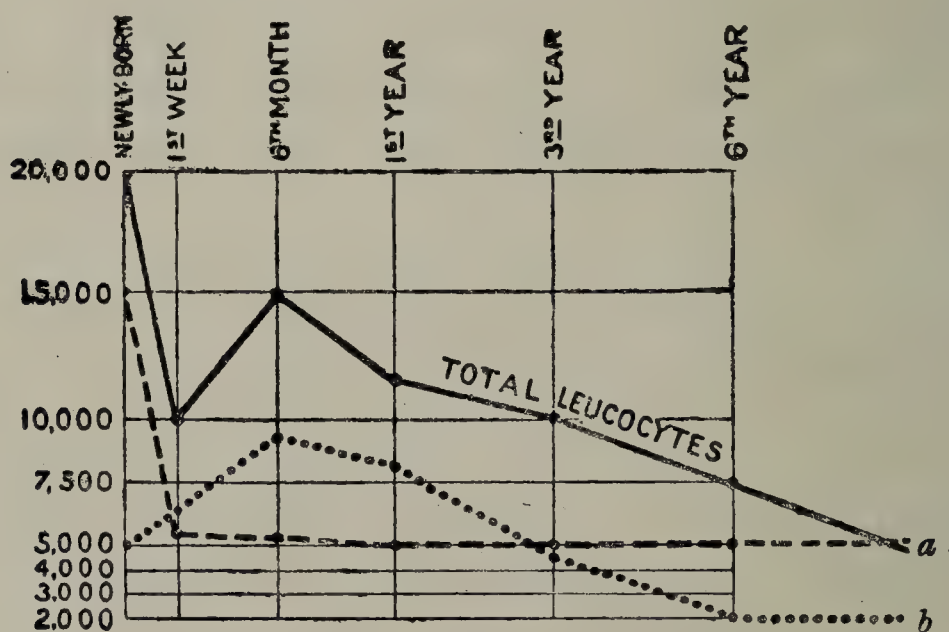


FIG. 140.—Absolute number of Leucocytes per cubic millimetre at different ages. *a*, Polynuclears; *b*, Lymphocytes. (R. Hutchison.)

non-granular leucocytes and the relatively low percentage of hæmoglobin, and in estimating the colour index during childhood it is important to remember this fact.

The blood platelets number 200,000 to 400,000 per cubic millimetre.

The bleeding time, clotting time, and fragility of the red-blood corpuscles are as in the adult.

Leucocytosis.

In childhood, as in later life, the condition of the blood as to leucocytosis is sometimes of great assistance in the diagnosis of septic and inflammatory states as well as in blood diseases. It is also of some value in prognosis under certain circumstances.

In a few diseases, such as tuberculosis, influenza, mumps, measles, r  theln, enteric, and malaria, there is usually no increase of the leucocytes *in uncomplicated cases*. In many cases of tuberculous meningitis, however, there is a very considerable increase. In nearly all the other infective diseases, and in most inflammatory affections, there is a decided polymorphonuclear leucocytosis.

The leucocyte count may throw light on the diagnosis of many other obscure conditions. It may help us, for example, in distinguishing a case of typhoid fever from one of appendicitis, septic  mia, or purulent meningitis, or an appendicitis from renal colic, acute indigestion, or enteric fever.

The absence of the usual leucocytosis in an infectious disease may be due to the attack being a mild one, or to the patient's low vitality. In the latter case it implies a grave prognosis. In a severe attack of pneumonia a high degree of leucocytosis is usually considered of good omen.

A leucocytosis in which the *lymphocytes* predominate is characteristic of *whooping-cough* and *glandular fever*. In whooping-cough the white cells may number as many as 85,000 per cubic millimetre and 80 to 90 per cent. consist of mononuclears. This absolute lymphocytosis is often of help in the diagnosis of a doubtful case of pertussis. In glandular fever an increased white count appears soon after the enlargement of the glands is apparent: at first this is due chiefly to a polymorphonuclear increase, but later it is the mononuclears which predominate. Tidy¹ records during the second week of illness a leucocytosis of 33,000 per cubic millimetre, of which 95 per cent. were lymphocytes. Both of these causes of lymphocytosis must always be borne in mind when considering the diagnosis of leuk  mia.

¹ H. L. Tidy, *Proc. Roy. Soc. Med.*, 1931, xxv. (Sect. Med. 1).

Eosinophilia.—In children, as in adults, eosinophiles constitute from 2 to 4 per cent. of the leucocytes in normal blood. A decided increase in their number is caused by the presence of animal parasites in the bowel or elsewhere. Eosinophilia is also met with in certain skin diseases, such as urticaria pigmentosa, dermatitis herpetiformis, and various bullous eruptions. An increase also occurs in asthma and other morbid conditions depending on anaphylaxis.

ANÆMIA.

Although anæmia is not uncommon during infancy and childhood, one must always be on his guard in making such a diagnosis, as many pale and feeble-looking children are not suffering from bloodlessness. The colour of the skin and conjunctivæ is often misleading. The colour of the lips is a better guide, but for a correct diagnosis a blood examination is essential. The pallor of the skin is often a vasomotor phenomenon due to different causes. If dyspepsia should be the underlying factor, the administration of iron will further damage the digestion and do more harm than good. Such cases are rather benefited by an ordinary alkaline or bitter tonic. On the other hand, it must be remembered that dyspepsia is often secondary to anæmia.

One of the most striking features of the anæmia of infancy and childhood is the evidence in the blood of the most intense regenerative changes from the greatest variety and often the slightest of causes. In contrast to what occurs in the adult, megalocytes and megaloblasts and myelocytes are of the commonest occurrence. This is no doubt due to the younger age of the patients, so that there results more easily a reversion to the embryonic type of the hæmatopoietic tissues. While in the adult this type of reaction is always associated with the most serious and often fatal varieties of anæmia, in the child it possesses no special significance. It is also because of the general tendency to this type of blood change that the classification, and also the prognosis, of anæmia during the early years of life are so difficult.

(A). Congenital Anæmia.

Apart from the polycythæmia which is characteristic of congenital pulmonary stenosis and some other cardiac malformations, no congenital abnormalities of the blood have been

generally recognised. Examples of severe anæmia developing during the first days of life might at first sight be considered possibly of congenital origin, but as a rule they have been found to be dependent on some post-natal cause, *e.g.*, internal hæmorrhage. The blood changes are characteristic of secondary anæmia.¹

Congenital Family Cholæmia (acholuric jaundice), however, undoubtedly belongs to this category, depending as it does on an hereditary increased fragility of the red-blood corpuscles. As already mentioned when discussing this malady in detail (p. 366), this peculiarity of the red cells, tending to their too ready destruction, also occurs occasionally as a non-hereditary phenomenon.²

(B). Primary Anæmia.

(1) **Chlorosis.**—Occasionally we see older children who are suffering from a type of anæmia which closely resembles chlorosis. There is no obvious cause for the bloodlessness, but the children are extremely pale and often have hæmic murmurs; there is little or no enlargement of the spleen. The blood shows a small percentage of hæmoglobin, with little diminution in the number of the red cells. The lymphocytes may be somewhat increased, the polymorphs being diminished in number. Severe cases of this type of anæmia are uncommon, but occasionally the children look distinctly ill and a considerable amount of œdema may even be present. The treatment is most satisfactory, as the child rapidly and completely recovers when some preparation of iron is given.

(2) **Pernicious Anæmia.**—No undoubted example of pernicious anæmia has ever been recorded in the child. However, as already indicated, a blood picture simulating that of Addison's anæmia is not unusual, but, from the fact that the majority make a complete recovery, it is evident that the blood picture *per se* cannot be taken as the criterion for diagnosis. These experiences also indicate that, in judging of the outlook of an example of anæmia, it is well never to be in a hurry to form a bad prognosis on the strength of the blood picture alone, but always to wait until sufficient time has elapsed to watch the effect of treatment. Some infants, who look wonderfully well,

¹ E. Pritchard and J. Smith, *Arch. Dis. Child.*, 1931, vi., 313.

² F. Parkes Weber, *Proc. Roy. Soc. Med.*, 1930, xxiv. (Clin. Sect. 1).

show a very alarming blood picture, and yet they make a good recovery. In other cases, although the blood film is not at all characteristic, the degree of cachexia strongly suggests a grave anæmia and their subsequent progress seems to confirm this diagnosis, or shows at least that they are instances of aplastic anæmia or perhaps some other variety of grave anæmia (Thursfield).

(3) **Leukanæmia.**—Leube¹ in 1900 saw in a boy what he took to be a combination of pernicious anæmia and leukæmia. Examination of the blood revealed a high colour index, no doubt due to the degree of megalocytosis present. The white cells numbered 10,600 per cubic millimetre, and of these 13 per cent. were neutrophil myelocytes and 0.6 per cent. eosinophilic myelocytes. At the autopsy there was neither pernicious anæmia nor leukæmia. We have already seen that megalocytosis with a high colour index and the presence of embryonic white cells (myelocytes) are common occurrences in any anæmia during childhood. This shows the danger of creating a new disease simply on the presence of an unusual number of cells in the circulating blood and without any histological basis.

(4) **Sickle-Cell Anæmia.**—This is a type of primary anæmia which has been described within recent years and is so-called because of the sickle-shape of the red-blood corpuscles. The cause is unknown. It is a familial disease and is confined to individuals of negro race or those of mixed blood. There is a moderately severe degree of anæmia, the red cells varying from 3,000,000 to 1,000,000 per cubic millimetre with the hæmoglobin reduced in proportion, and a leucocytosis of between 20,000 and 60,000 per cubic millimetre. The diagnosis rests on the discovery of the peculiar shape of the red cells, which may be affected to the extent of 20 to 30 per cent. of the total number. The course of the disease is very prolonged; it may last many years, and there has not as yet been discovered any specific therapy.

(C). Secondary Anæmia.

Most of the anæmias of childhood are of this nature. There is a diminution in the number of the red-blood corpuscles with a relatively greater fall in the hæmoglobin, resulting in a low colour index, and a varying degree of leucocytosis. Nucleated

¹ O. Naegeli, *Blutkrankheiten und Blutdiagnostik*, Berlin, 1931, 346.

red-blood corpuscles and various types of degeneration (polychromatophilia and basophilia) are not uncommon.

Secondary anæmia may occur at any age, and from the greatest variety of causes. Hæmorrhage at birth, internal hæmorrhage during the early days of life, as well as the hæmorrhagic disease of the new-born, are the common causes in the very young infant. During later infancy a deficient intake of iron through feeding with dilute milk mixtures or a too prolonged milk diet, and a diet deficient in vitamin C with the development of scurvy, are the causes to be borne in mind. During later childhood the causes are many. Infectious diseases of any kind are apt to cause it, and the rheumatic poison is specially prone to do so. Tuberculosis, syphilis, malaria, chronic diarrhœa, coeliac disease, and septic conditions from bad teeth may all lead to severe secondary anæmia. The particular etiological factor causing the anæmia in the various conditions is difficult to assess. In all, defective nutrition and toxic action both play a part. The same is true of the so-called "school anæmia," as probably both hygienic and nutritional factors take part in its production. Poisoning by minerals such as lead and arsenic, although rare in childhood as a cause of anæmia, does occur. Anæmia from lead poisoning may develop even in the breast-fed infant from the mother wearing lead nipple-shields or applying lead lotions to the mammæ. Any severe degree of basophilia should always raise the question of lead poisoning. In the older child anæmia may result from the prolonged administration of arsenic, but as this usually happens in a rheumatic patient it is difficult to say whether the arsenic or the rheumatic poison is the chief etiological factor.

Von Jaksch's Anæmia.—(Splenic Anæmia of Infancy—Anæmia Infantum Pseudoleukæmica).—In former years this was considered not only the most characteristic but most common blood disease of infancy. However, it has been observed much less frequently within recent times, and, like chlorosis, is generally stated to be disappearing. Although without doubt one seldom nowadays sees the marked examples of former days, this is more likely due to our better methods of diagnosis and the better classification of the blood diseases and splenomegalies at this period of life than to the disappearance of a definite clinical entity. In fact, it is doubtful if

von Jaksch's anæmia is a disease *sui generis*.¹ Von Jaksch described the condition in the first instance on the strength of only two examples, and it is interesting to note that one of these was shown in the post-mortem room to be a case of lymphocytic leukæmia. At one time the disease was reported to be not uncommon in Italy and the South of France along the shores of the Mediterranean, but it was later demonstrated that the cause of this variety of splenic anæmia was infection with the Leishman-Donovan bodies (Anæmia Leishmania).² It is also not improbable that some of the earlier and fatal cases were examples of Gaucher's disease.

The features which go to make up the clinical syndrome of this disease are a varying degree of secondary anæmia with leucocytosis, the presence of nucleated red-blood corpuscles and myelocytes, and an enlarged spleen in a child under three and a half years of age. But, as we have already seen, the presence of nucleated red cells and myelocytes, as also an enlarged spleen, are just the very changes which are apt to be present in any severe anæmia at this age period, and the opinion is steadily growing that von Jaksch's anæmia is not a specific disease but is simply the infantile type of reaction to any agent which is causing a severe anæmia. The conditions under which it was met in this country support such a view. It was prone to occur in badly-nourished children, living under unhygienic conditions; rickets was frequently present, and also syphilis, but it was noted in well-to-do families.

Splenic Anæmia (*Banti's Disease*).—This disease, although chiefly an affection of adult life, is occasionally met with in childhood and even early infancy. We ourselves have observed an undoubted example in an infant of eight months, in whom the illness dated from the age of six months. The disease is characterised by anæmia with a low colour index and leucopœnia. After a variable period splenic enlargement appears, and to judge from personal experience, sooner in the child than in the adult. At a still later date cirrhosis of the liver develops, with ascites and gastric hæmorrhage. By some writers the term "Banti's Disease" is reserved for this final stage, but it is only fair to Banti to say that he pointed out that, if the spleen (in

¹ G. W. St C. Ramsay, *Brit. Journ. Child. Dis.*, 1924, xxi., 48.

² Cannata, *Pediatrics*, May 1910, and *Revue Générale, Arch. de Med. des Enfants*, 1910, xiii., 932.

which he thought there was elaborated a toxin which led to the anæmia and other sequelæ) were removed, cirrhosis of the liver would not develop.

The patient may come under observation on account of lassitude, increasing pallor, questionable jaundice, and gastrointestinal trouble, but not infrequently a sudden attack of hæmatemesis may be the cause of advice being sought. In some of these latter cases it may be stated that the child has always been delicate and pale and perhaps has bruised easily.

The child is almost without exception anæmic; the skin has a lemon-yellow tint and the spleen is found enlarged. The enlargement of the spleen may be extreme, the lower edge extending six or seven finger-breadths below the costal margin, and the anterior edge to the right of the umbilicus. As a rule the liver is not palpable, or at least only so in the epigastrium, where in later stages of the disease it may be felt somewhat nodular. If the disease is far advanced there may be free fluid in the peritoneal cavity. A slight purpuric eruption may be present, and a variable degree of fever.

Examination of the blood will reveal, as a rule, a severe degree of anæmia—the red cells varying between 1,000,000 and 2,000,000 per cubic millimetre, and the hæmoglobin between 20 and 30 per cent. The leucocytes are invariably diminished and may not number more than 2500 per cubic millimetre: stained films seldom show much abnormality beyond some variation in the size of the reds and occasionally the presence of a proportion of myelocytes. Nucleated red cells are never present.

Hæmic murmurs are frequently audible. The urine on rare occasions may contain a trace of urobilin, but Van den Bergh's test of the serum is usually negative. The red blood corpuscles reveal a normal degree of fragility, and Wassermann's test is negative.

Gaucher's Disease.—Although this affection is not always accompanied by anæmia, this is the case so frequently and since the malady presents so many similarities to Banti's disease, as well as to other blood diseases with splenomegaly, it is appropriate that it should be considered here.

Gaucher's disease is a disorder of metabolism in which the cells of the reticulo-endothelial system throughout the body

(spleen, adenoid tissue, liver, and bone) are infiltrated with some abnormal or unwanted lipoid product.^{1, 2} The type of lipoid present is not always of the same constitution. Niemann-Pick's disease is undoubtedly an allied condition.

Gaucher's disease is definitely familial, as several members in a family may be attacked. It is impossible to say at what age the disease first appears. In onset it is usually insidious. It may, however, give rise to no symptoms and the discovery of the splenic tumour is accidental. On occasion, enlargement of the abdomen, or pain and a feeling of dragging in the left side from increase in size of the spleen, are the cause of drawing attention to the condition. When the long bones are affected a pathological fracture has first attracted attention.

The characteristic feature of the condition is an enlarged spleen. This may be extreme, and in the infant amount to one-sixth of the body weight. Although the disease invariably attacks the glands, and post-mortem those of the abdomen and thorax are usually found enlarged, the superficial glands are not as a rule palpable. A moderate degree of enlargement of the liver is almost constant, which is an important differentiating feature between this disease and Banti's anæmia.

Examination of the blood may reveal a degree of anæmia as extreme as is met with in any example of Banti's disease. This is not the rule, however, and many cases give normal red-cell and hæmoglobin estimations. There is, however, a constant *leucopænia*—this is an *invariable feature* and its absence should always make one hesitate to diagnose Gaucher's disease. Nucleated red cells are never present, and the fragility of the hæmocytes is normal. Myelocytes are always present. A tendency to hæmorrhage is common and may occur from the gums, nose or lips, or into the skin. True jaundice has been observed occasionally.

The course of the illness is chronic and extends over many years. There is no known treatment, but if the size of the spleen is giving rise to much discomfort, which not infrequently happens, or if the anæmia with leucopænia is marked and thus evidence of hypersplenism,³ splenectomy may be performed with relief.

¹ S. Graham and J. W. S. Blacklock, *Arch. Dis. Child.*, 1927, ii., 267.

² A. Moncrieff, *Arch. Dis. Child.*, 1930, v., 265.

³ F. Parkes Weber, *Brit. Med. Journ.*, 1929, i., 766.

Differential Diagnosis of Anæmia with Splenomegaly.—

It will have been apparent from the various descriptions of the different types of anæmia with splenomegaly given above that the differential diagnosis must at times be a matter of extreme difficulty. Not only may Gaucher's disease, Banti's disease, von Jaksch's anæmia, and acholuric jaundice be confused with one another, but syphilis, tuberculosis, cirrhosis of the liver with or without implication of the pancreas and spleen, as well as leukæmia, thrombosis of the splenic vein, and Hodgkin's disease can all give rise to an almost identical clinical picture. The possibility of confusion of these various pathological states is strikingly revealed in the Table on p. 478, in which the incidence of the various findings is shown.

In the first place, the blood picture should reveal if the case is one of leukæmia, and a leucocytosis would be in favour of either Hodgkin's disease or acholuric jaundice. In all the other conditions a leucopœnia is invariable. Nevertheless, an example of aleukæmic leukæmia, *i.e.*, one in which there is no increase of the leucocytes, might present difficulty; but the fact that the white cells were of embryonic type, and that there was an absolute increase in the total number per cubic millimetre of one variety (lymphoid or myelocytic) of cell, would decide the issue. In acholuric jaundice and Hodgkin's disease, too, a leucocytosis is not invariable.

The history of recurrent attacks of illness characterised by pallor, fever, jaundice, and abdominal pain, with a possible family history of the same type of condition, should suggest acholuric jaundice. This diagnosis is confirmed by detecting increased fragility of the red cells, which is peculiar to this disease and not found apart from it. Examples of hæmolytic jaundice exactly similar in their behaviour to acholuric jaundice, but in which there is no increased fragility of the red cells, are occasionally met with.¹ It would appear that in these cases the toxin, or whatever is the cause, has the ability to destroy the normally resistant cells.

Thus, by a process of exclusion, leukæmia, acholuric jaundice and Hodgkin's disease are eliminated, and the possibility of Gaucher's or Banti's disease, or one of the rarer types of hepatic cirrhosis, is left. A diagnosis of Gaucher's or Banti's disease, or hepatic cirrhosis, should, however, never be made in the absence

¹ F. Parkes Weber, *Proc. Roy. Soc. Med.*, 1931, xxv. (Clin. Sect. 9).

Table showing Differential Diagnostic Features of Anæmia with Splenomegaly.

Disease.	Familial History.	Splenomegaly.	Enlargement of Liver.	Blood.						Hæmorrhages.	Fever.	Diagnostic Value of Splenic Puncture.
				Anæmia.	Leucopœnia	Nuc. Reds.	Myelocytes.	Fragility of Reds.	W. R.			
Gaucher's Disease ¹	+	+	+	+ or -	++	+	++	+	+	++	++	++
Banti's Disease	+	+ or -	+	++	++	+	++	+	+	++	++	++
Acholuric Jaundice	-	+ or -	+	++	+	+	+	+	+	+	+	+
Cirrhosis of Liver ²	+ or -	+	+	+	+	+	+	+	+	+	+	+
Spleen and Pancreas.	+	+	+	+	+	+	+	+	+	+	+	+
Thrombosis of Splenic Vein ³	-	+	+	+	+	+	+	+	+	+	+	+
Hodgkin's Disease.	-	+	+	+	+	+	+	+	+	+	+	+
Leukæmia (Aleukæmia).	-	+	+	+	+	+	+	+	+	+	+	+
Tuberculosis of Spleen	-	+	+	+	+	+	+	+	+	+	+	+
Syphilis ⁴	+	+	+	+	+	+	+	+	+	+	+	+
Splenic and Pancreatic Cirrhosis ⁵	-	+	+	+	+	+	+	+	+	+	+	+

¹ S. Graham and J. W. S. Blacklock, *Arch. Dis. Child.*, 1927, ii., 267; A. Moncrieff, *Arch. Dis. Child.*, 1930, v., 265.
² F. D. Gunn, *Arch. Path. and Lab. Med.*, 1926, i., 527, and personal observation (L. F.).
³ J. Kretz., *Med. Klinik.*, 1929, xxv., i., 299.
⁴ W. Osler, *Clin. Journ.*, 20th July 1914, 462, and personal observation (L. F.).
⁵ Personal observation (L. F.).

of Wassermann's test for syphilis. Osler¹ has described examples of congenital and acquired syphilis reproducing exactly the clinical picture of Banti's disease, and this we have seen ourselves on two occasions in the case of congenital lues. As often happens in the case of syphilis tarda, no history of early manifestations is obtained and there is a complete absence of all stigmata, the positive Wassermann reaction being the only evidence of the infection. Between Banti's anæmia and Gaucher's disease a familial history, some enlargement of the liver, an absence of anæmia and absence of fever would be in favour of the latter malady, though enlargement of the liver may be present in Banti's disease and is the rule in the anomalous examples of cirrhosis of the liver with or without involvement of the pancreas and spleen, in which, too, a familial tendency has been described.² An earlier age of onset is also rather in favour of Gaucher's disease, although we have observed what appears to be typical Banti's disease in an infant of eight months. The only definite criterion for diagnosis in these diseases is, however, the histological examination of the affected organs; in fact, without such an examination the diagnosis always remains problematical. For this purpose splenic puncture, and hepatic puncture also if it is thought desirable, is of the greatest assistance. This operation would seem to be devoid of all danger and is easily carried out. Into the spleen, firmly grasped in the left hand and pushed against the lower ribs and abdominal wall, a needle of fairly wide bore (No. 15), with syringe attached, is inserted in much the same way as is done when sampling cheese. A strong negative pressure is maintained in the syringe while the needle is withdrawn, and any blood and tissue obtained mixed with a few cubic centimetres of distilled water to lyse the corpuscles. The whole is then centrifugalised and the sediment passed through 5 per cent. and then 10 per cent. formalin and fixed in paraffin, from which serial sections are cut and examined. In this way perfect pictures of Gaucher's disease, Banti's disease, and tuberculosis of the spleen may be obtained (Figs. 141 to 143). Direct smears of the material removed by puncture may be made and these have on occasion revealed the presence of the Gaucher cells, but this method is less

¹ W. Osler, *Clin. Journ.*, 1914, xliii., 462.

² F. D. Gunn, *Arch. Path. and Laby. Med.*, 1926, i., 527.

satisfactory than sections and is, of course, of no assistance in the recognition of fibrotic changes.

Treatment of Anæmia.—The correct treatment naturally depends on a correct diagnosis. In acholuric jaundice, as already mentioned, splenectomy is called for. Although this operation does not modify the true cause of the disease, viz., the increased fragility of the red-blood corpuscles, it removes the agent which brings about their destruction. In Banti's disease, if recognised early, splenectomy is also advisable. Excision of the spleen we have seen performed in von Jaksch's anæmia, but it did not

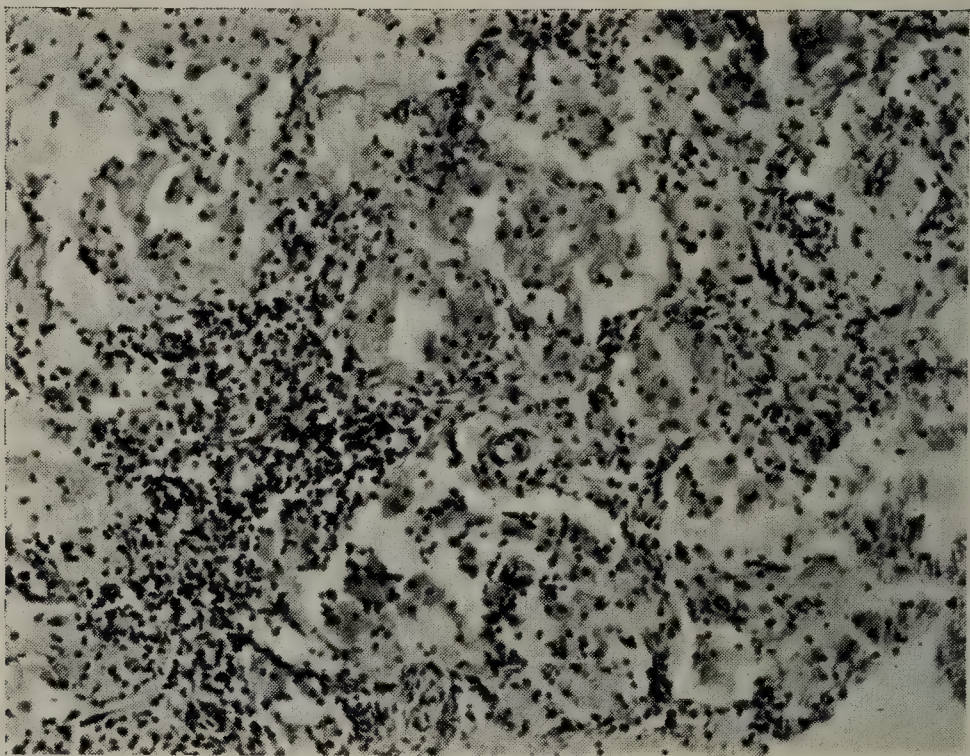


FIG. 141.—Section of piece of tissue obtained by splenic puncture, showing typical appearance of Gaucher's disease. Note the alveolar arrangement of "foamy" epithelioid cells. $\times 140$.

seem to modify in the least the course of events. Otherwise the treatment of anæmia is governed by general principles.

If the degree of anæmia is severe a blood transfusion should be carried out after blood grouping tests have revealed a satisfactory donor, whether it is a case ultimately suitable for surgical or medicinal treatment. If the donor has not been suitable, alarming symptoms may appear—hæmolysis with bilirubin jaundice and hæmoglobinuria. In many severe examples of anæmia this procedure alone is able to save the child, and in certain cases it would appear to be the only true stimulus to regeneration, so that no case should be considered hopeless until this method of treatment has been given a trial. One transfusion is often sufficient to turn the balance, but in

some cases two or even three may be required before satisfactory recovery sets in. Fifteen cubic centimetres per pound body

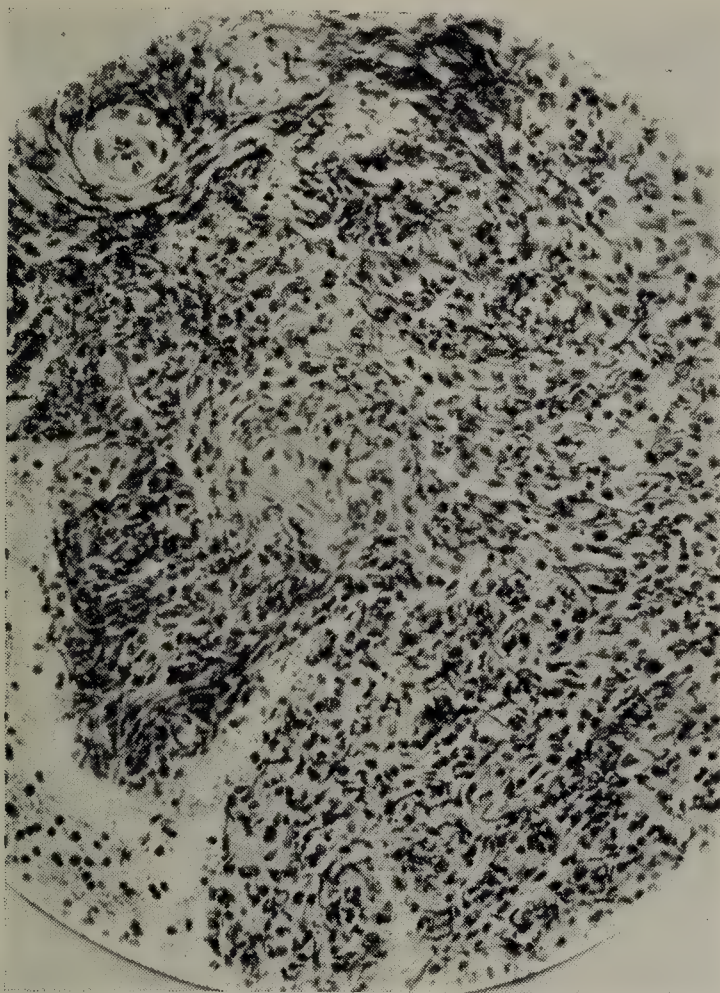


FIG. 142.—Section showing Fibrosis of Spleen in piece of tissue obtained by splenic puncture. $\times 200$.

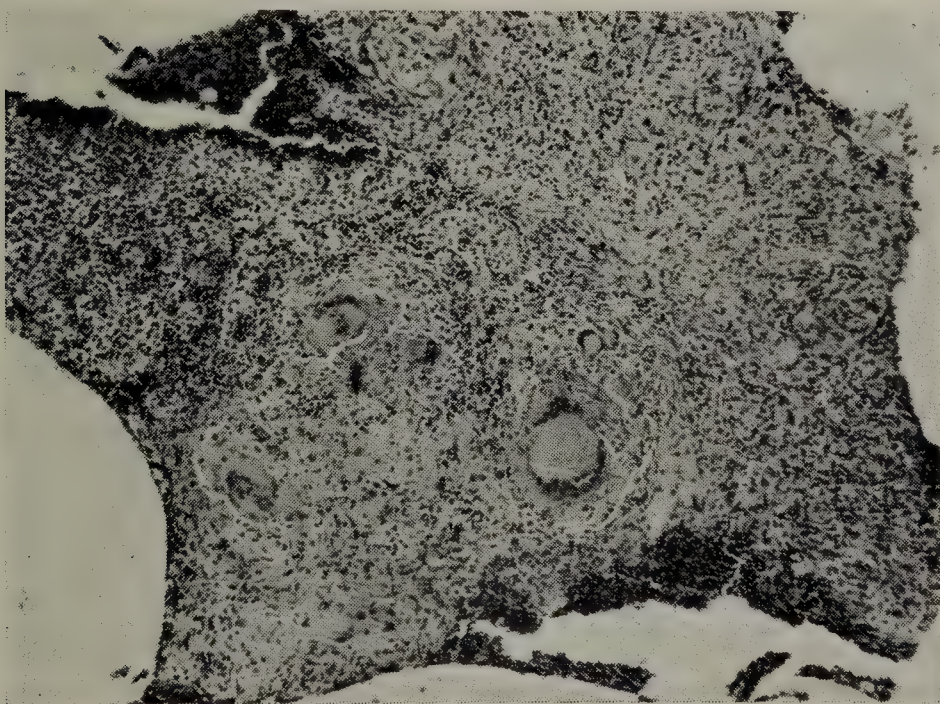


FIG. 143.—Section showing typical Tuberculosis in piece of tissue obtained by splenic puncture.

weight is the maximum amount of blood which should be given at any one time. For the technique of blood transfusion, see p. 1014

If the anæmia is moderate in severity some medicinal preparation of iron (Blaud's pill, one t.i.d.; vin. ferri. cit. 3ss. to 3i. t.i.d.; ferri. carb. grs. v. t.i.d.; ferri. et ammon. cit. grs. v. t.i.d.) alone or in combination with arsenic (liq. arsenicalis min. i. to iii. t.i.d.) should be tried in the first instance. In our experience all preparations of iron are equally efficient, but there is no doubt that the combination of iron and arsenic gives better results than are got with iron alone. On occasion neither iron nor the combination of iron and arsenic seems to have any effect, and in these circumstances extract of liver should be given a trial, as good results have been recorded. At present it is impossible to recognise the special conditions calling for one or the other variety of medicinal treatment, and they must simply be tried in rotation. As above noted, however, no case should be considered refractory without resort to transfusion.

The diet, the hygienic conditions, and any contributory condition recognised (sepsis, tuberculosis, etc.) should be attended to.

Leukæmia.

Leukæmia during infancy and childhood may be either lymphocytic or myelocytic. These terms are employed in preference to lymphatic and myelogenous because it is now generally admitted that the pathological process is always primarily situated in the bone-marrow, whereas the older nomenclature suggests a double origin from the lymphoid and myelogenous tissues.

Of 100 examples of leukæmia occurring during childhood analysed by Ramsay,¹ 70 per cent. were lymphocytic and 19 per cent. were myelocytic in nature. Occasionally examples of the disease occur in which the prevailing leucocyte circulating in the blood is of a very early type, and has not become differentiated into either a lymphocyte or myelocyte. This variety of leukæmia is called myeloblastic or, preferably, lymphoidcytic. This behaviour of the white cells when undergoing active multiplication is in keeping with the marked tendency to the appearance of very early or embryonic forms (myeloblasts) of the red-blood corpuscles in anæmia during early childhood. It is the appearance of these same embryonic

¹ G. W. St C. Ramsay, *Arch. Dis. Child.*, 1927, ii., 119.

types of leucocytes in the myelocytic form of the disease, or the appearance of a certain proportion of the more mature and granular forms in the lymphoidocytic type, which is responsible for the conception of mixed leukæmia, *i.e.*, a leukæmia in which both the adenoid and myeloid tissues are implicated. A true mixed leukæmia, however, does not occur.

An increase in the number of the white cells is the chief characteristic of the disease. Sometimes, however, there is no absolute increase in the number of white cells circulating in the blood. Indeed, there may be a leucopœnia, but of the white cells present the majority are abnormal or embryonic in nature and thus discloses the true nature of the mischief. When this occurs the condition is called *atypical* or *aleukæmic leukæmia*. In this connection it cannot be too strongly emphasised that the acute-ness or severity of the malady is not reflected in the number of white cells in the blood stream. Very acute cases, and cases towards the termination of the disease, tend to show either no increase or only a very slight increase in number; on the other hand, in the aleukæmic cases the white cells frequently increase in number rapidly towards the end.

In the above-mentioned series of 100 examples of leukæmia reviewed by Ramsay, the disease occurred twice as frequently in children under four years of age as in those over four years. In some of the children the condition was recognised at as early an age as six weeks, and in a few the symptoms dated from birth, so that the question of a congenital origin is raised. Boys were twice as often affected as girls.

In general, the symptomatology varies little whatever the type of leukæmia. The disease may be insidious or sudden in onset. When gradual in onset the features are lethargy, anorexia, loss of weight, and increasing pallor. Such prodromal symptoms may persist for two or three months, but sooner or later hæmorrhages, splenomegaly, and glandular enlargement make their appearance and suggest a blood examination, which will reveal the true nature of the illness. When the onset is sudden it is usually by the development of fever, great prostration, and purpuric manifestations. At this stage there may be neither glandular nor splenic enlargement, and unless a blood examination is made the nature of the malady will be overlooked.

Although splenic enlargement is present in over 90 per cent.

of the cases when the condition is well advanced, splenomegaly is so often present from other causes that it is *per se* of little value in the diagnosis. Enlargement of the glands is seldom extreme and may be absent. Those in the neck are the ones usually involved. Hæmorrhage into the skin or from the mucous membranes (nose, gums, stomach, and bowel) or into the retina, is a common feature, and should always suggest the possibility of the patient suffering from leukæmia. This is more particularly the case if accompanied by fever or splenic or glandular enlargement.

Leukæmia during infancy and childhood is essentially an acute disease. In all types of the disease the duration is seldom more than two or three months.

The following are the blood changes observed in the various types of the disease.

(a) *Lymphocytic leukæmia*.—The total red cell count is always decreased and may fall to less than one million per cubic millimetre. Poikilocytosis, polychromatophilia, and basophilia are common features. The leucocytes are as a rule much increased—200,000 or more per cubic millimetre—although occasionally the white cell count is either normal or decreased. Over 90 per cent. of the white cells are lymphocytes: the commonest type of lymphocyte is the small or moderately sized lymphocyte, but in some cases, and especially in the acuter varieties, the large lymphocyte predominates. It must be remembered, of course, that in whooping-cough and glandular fever just as great a relative and absolute number of lymphocytes occurs (p. 469).

(b) *Myelocytic leukæmia*.—Of the nineteen examples analysed by Ramsay the average red cell count was 2,114,800 per cubic millimetre and the hæmoglobin content 31.8 per cent., giving a colour index of 0.76. The average white cell count was 139,020 per cubic millimetre, with an average proportion of myelocytes amounting to 32 per cent. Ramsay supplies the following differential count of the white cells:

Neut. Polymorphs . . .	38.7 per cent.
Eosin. „ . . .	3.6 „
Lymphocytes . . .	25.0 „
Myelocytes . . .	32.0 „

(c) *Lymphoidocytic or myeloblastic leukæmia*. The average red cell content in eleven cases was 2,066,400 per cubic millimetre,

and the average hæmoglobin content 31 per cent., giving a colour index of 0.77. In this variety of the disease the increase in the white cells is distinctly less than in either the lymphocytic or myelocytic varieties. Of the cases analysed the white cell count averaged 47,962 per cubic millimetre; in two cases there was a leucopœnia.

The average differential leucocyte count in these cases was—

Neut. Polymorphs	9.3 per cent.
Eosin. „	2.0 „
Lymphocytes	22.5 „
Myelocytes	4.6 „
Lymphoblasts or Myeloblasts	58.0 „

The *treatment* of leukæmia is most unsatisfactory. Temporary improvement sometimes follows the use of increasing



FIGS. 144 and 145.—Chloroma. (Boy of 5 years.) (Dr Melville Dunlop's case.)

doses of arsenic along with fresh air and careful dieting and nursing. The application of X-rays causes a striking diminution in the size of the spleen, but it has no permanently good effect on the disease.

(*d*) *Chloroma*.—Chloroma is a rare form of leukæmia, which may be either myelocytic or lymphocytic in nature. It is characterised by the formation of tumours of a peculiar greenish colour, which are found chiefly in the orbits or on other parts of the skull, over the long bones, and in certain of the viscera. The tumours have the histological characters of either adenoid or myeloid tissue.

The blood has the features of leukæmia, but the blood picture is often atypical. There may be no increase in the total number of white cells and hence is of the aleukæmic variety. The cells are for the most part embryonic and large in the lymphocytic variety, and myeloblastic in nature when the disease is of the myelocytic type.

In typical cases it gives rise to a very characteristic appearance of the face, which is well seen in Figs. 144 and 145. A considerable proportion of the reported cases have occurred in children. It is quite incurable and the illness lasts for six to eight weeks. The orbital swellings are sometimes mistaken for sarcomata, or for the metastases of malignant medullary hypernephromata (p. 521). The condition may also be mistaken for myeloma, a malignant tumour of the bone-marrow which may cause the appearance of a proportion of 10 to 20 per cent. of myelocytes in the circulating blood.

Purpura.

Purpura is a common symptom in childhood, and, as in adult life, it occurs under a great variety of conditions. It may be either symptomatic or idiopathic.

(a) **Symptomatic Purpura.**—In young children purpuric symptoms are met with in cases of severe jaundice from any cause, in scurvy, in leukæmia, and in various other forms of anæmia with enlargement of the spleen, in syphilis, in miliary tuberculosis, in septic conditions of many kinds, and in severe types of most of the acute infective diseases, including rheumatism and epidemic cerebro-spinal meningitis. They also occur in poisoning with various drugs, notably iodide of potassium in susceptible individuals, and in cachectic conditions generally, whether due to diarrhœa, atrophy, or other diseases.

A form of purpura has been described which is due to the action of hæmolytic bacteria. The symptoms may closely resemble those of Henoch's purpura.

The purpura which accompanies severe diarrhœa is of especial interest. It is not at all uncommon in cachectic children under twelve months. The front of the abdomen and the lower part of the thorax are the parts mainly affected. It is a late, and usually an ominous symptom. We have, however, had several cases which recovered although the purpuric eruption had been extensive.

Subcutaneous hæmorrhages from mechanical causes are sometimes met with in cases of epilepsy, and they are common in whooping-cough, especially in the eyelids and under the conjunctivæ.

The *treatment* of symptomatic purpura is that of the original disease.

(*b*) **Idiopathic Purpura ; Primary Purpura ; Hæmorrhagic Diathesis.**—In this condition there occurs spontaneous extravasation of blood into the skin and from the mucous membranes. Although the exact cause is unknown, it is believed to be due to an increased permeability of the capillary endothelium, much in the same way as plasma escapes in the urticarias. It is worthy of note that in some cases both urticarial and purpuric phenomena are present at the same time.

Because the blood platelets are much diminished in some of the examples of purpura, and especially if the hæmorrhages are severe, it has been suggested that in some way their diminution is the essential cause, and hence the term *thrombocytopenic purpura*. However, when the endothelium of the blood vessels is injured in any way, the platelets deposit at the seat of injury and offer protection against the escape of the blood, so that it is quite possible that the thrombopœnia is a secondary phenomenon. In support of this view are the facts that not infrequently in some cases hæmorrhage ceases although the platelets remain diminished, and in other cases the hæmorrhage continues in spite of a normal number of platelets.

Removal of the spleen was noted by Kaznelson¹ to benefit some cases of purpura, and in consequence he developed the theory that the spleen produced a toxin which destroyed the platelets. As noted above, however, there is no direct evidence that thrombopœnia is the real cause of the condition, and Tidy² remarks that the splenic toxin, if such there be, may rather act on the capillary endothelium and increase its permeability. But against the spleen playing any part at all is the fact that the purpura may recur, and that even comparatively soon after splenectomy. This occurrence has been explained by postulating that the reticulo-endothelium system takes on the function of the spleen and produces the toxin.

The dominating feature of the condition is hæmorrhage

¹ P. Kaznelson, *Wien. Klin. Wochensch.*, 1916, xxix., 1451.

² H. L. Tidy, *Brit. Med. Journ.*, 1930, II.

from the skin or mucous membranes. The hæmorrhage may be widespread but at times a single mucous membrane (nose, kidney, uterus, or stomach) may be involved. In addition to the extravasation of the blood corpuscles in certain circumstances the plasma alone may escape, and it is this which accounts for the urticarial phenomena, the swelling of the joints, and the colicky abdominal pains.

The examples of this condition are classified for the purposes of clinical description into (1) *purpura simplex*; (2) *purpura hæmorrhagica*; (3) *Henoch's purpura*. Essentially they are varieties of the same pathological process, merely varying according to the severity, the seat of the hæmorrhage, and the extent to which the urticarial phenomena appear.

1. *Purpura simplex*.—In this the eruption is accompanied by very slight general disturbance, with or without articular pains, but by no hæmorrhages from internal organs and usually no rise of temperature; and the symptoms usually only last for a week or ten days. The disease is a trivial one, and the prognosis is always good.

2. *Purpura hæmorrhagica* (Morbus Maculosus of Werlhof).—In this, in addition to the skin lesions, there is bleeding from one or other of the mucous surfaces, or possibly intracranial hæmorrhage. There is often a slight degree of fever, probably always some enlargement of the spleen, and, as has been recently shown, a diminution in the number of the blood platelets. The severity of the disease varies greatly in different cases. A number of them die as the result of the hæmorrhages. The clinical type described as *purpura fulminans* probably merely represents this disease in its most severe and fatal form. Cases of suprarenal hæmorrhage in infants are sometimes mistaken for it.

Like *purpura simplex* it begins with a crop of petechiæ and bruise-like ecchymoses in various parts of the body, and occasionally with a sudden hæmorrhage from the nose or elsewhere. Many of the acute cases recover in a varying number of days or weeks, with or without treatment. In other apparently similar cases, fresh attacks of the same kind go on recurring at intervals of a month or of several months, and the hæmorrhages may be dangerously severe. Oozing from the gums is also often seen. Besides the epistaxis, there may be hæmorrhages from the stomach, bowel, kidneys, or vagina, or

into the brain or meninges. In other respects the child seems fairly well until symptoms of anæmia set in as the result of the repeated hæmorrhages. The small degree to which the patient's general health and comfort suffer is characteristic of this disease.

On examination of the *blood*, a degree of secondary anæmia is usually found which varies with the severity of the case; but the outstanding features are:—

(1) A great *diminution in blood platelets* or thrombocytes, the number varying from 40,000 to 5000 or none at all, instead of the normal counts of from 200,000 to 400,000, per cub. mm.

(2) *Prolongation of the bleeding-time*.—This is tested by pricking the ear and noting how long the bleeding lasts. In this disease the time varies from ten to twelve minutes; sometimes it may be twenty minutes or very much longer (although the coagulation time is normal). In health it should be from one to two and a half minutes. The bleeding-time is not prolonged in purpura simplex or Henoch's purpura.

(3) *Non-retraction of blood-clot*.—Normal blood clots readily *en masse* within twenty minutes, and within an hour the clot will have firmly contracted. In this disease the clot forms in the usual time, but no contraction occurs even after standing for hours, owing to the want of the blood platelets.

Treatment.—In any severe example of the hæmorrhagic diathesis a blood transfusion should be carried out, as this not only counteracts the anæmia but may arrest the bleeding. Short of blood transfusion the subcutaneous injection of whole blood, as is so successful in the hæmorrhagic disease of the new-born, may be tried.

Success has occasionally been obtained by the intravenous injection of a 1 : 1000 solution of adrenaline, and injections of T.A.B. for their anaphylactic effect have also been used. As the condition is not due to diminished coagulability of the blood, salts of calcium are useless.

As previously mentioned, splenectomy has been credited with being almost a specific cure for this disease. Many successes have been recorded, but also many failures. The bleeding often stops spontaneously when the patient seems almost *in extremis*, and hence it is not infrequently a matter of great difficulty in ascribing the result to any therapeutic measure. It may be laid down as an invariable rule that a blood transfusion should always be performed before having resort to splenectomy.

3. *Henoch's Purpura*.—This name is given to a type of purpura which is mostly seen in children, and is especially important owing to the frequency with which it is mistaken for intussusception and other forms of intestinal obstruction, as well as from its pathological interest. Cases in which the joints are swollen and painful with purpuric spots in the neighbourhood are often called Schönlein's purpura (*purpura rheumatica*).

Its main *symptoms* are acute abdominal pain, with tenderness over the colon, vomiting, and either bloody diarrhoea or constipation. There are also swelling and pain of the joints, subcutaneous hæmorrhages, and bleedings from mucous surfaces. Although all these clinical features are generally present, any of them may be either absent or so slight as to be readily overlooked. Two other points are important in the matter of prognosis, namely, that the attack is apt to be repeated after a varying interval; and that acute nephritis not infrequently occurs, and is sometimes fatal.

Owing to the severe abdominal symptoms and the slightness of the subcutaneous and other hæmorrhages, Henoch's purpura is frequently mistaken for acute appendicitis or intussusception. Generally, however, the diagnosis is not difficult, if the facts of the case have been carefully investigated. Occasionally the resemblance to intussusception is increased in a most perplexing way, owing to the occurrence of hæmorrhage into the intestinal wall which gives rise to a palpable tumour. Some cases have also been reported in which such an extravasation has led to a secondary intussusception.

The relations of this form of purpura to such diseases as angioneurotic œdema, urticaria, and erythema multiforme are close and interesting.¹

The *treatment* is mainly expectant, and consists chiefly in opium and the application of an ice-bag to the abdomen during the acute attacks. As the purpura in these cases may be secondary to a bowel lesion which results in septic absorption, it is desirable that special attention should be given to the state of the alimentary canal during the intervals between the attacks. The use of rectal injections of anti-streptococcal serum is sometimes beneficial in this as in other forms of purpura.

¹ W. Osler, "The Visceral Lesions of Purpura and Allied Conditions," *Brit. Med. Journ.*, 1914, i., 517.

Hæmophilia.

It is said that hæmophilia manifests itself before the end of the second year in from 60 to 70 per cent. of the cases; but it is a striking fact that hæmophilic infants do not seem to suffer more frequently than other babies from umbilical hæmorrhages, cephalhæmatomata, or melæna neonatorum. It is later, after circumcision, division of the frænum linguæ, or abrasion of the gums, that the tendency to bleeding first appears. The patients are always boys, and the disease is transmitted through the mothers only. A history of a family tendency to bleed excessively can be obtained in most cases. The joint affections of hæmophilia, which are among its most interesting clinical manifestations, have been already described (p. 88).

According to recent views,¹ "the essential defect of hæmophilic blood, which accounts for its delayed coagulability, is a diminution of the circulating prothrombin. The other two fibrin factors, antithrombin and fibrinogen, are present in normal amounts."

Although we have now more means at our disposal for stopping the hæmorrhages than formerly, the *treatment* of the tendency to bleed is most unsatisfactory. The main points are as follows:—

1. In patients who have shown this tendency, no operation should be performed that is not absolutely necessary, and every care must be taken to avoid an injury to the joints.

2. To increase the coagulability of the blood, calcium lactate may be given twice a week in doses of 15 gr. (Wright). The subcutaneous injection of horse serum has also been found useful.

3. When hæmorrhage occurs and is difficult to arrest, the handiest method to use is the application to the bleeding point of fresh blood from a healthy individual, along with long-continued mechanical pressure. Hurwitz and Lucas have found that, "because of its great hæmostatic properties and its thermostability, kephalin deserves an important place in the treatment of bleeding from external wounds." When applied locally it brings the hæmorrhage to an early arrest, although when given by mouth or in subcutaneous or intramuscular injections "it has no effect on the disease process."

¹ S. H. Hurwitz and W. P. Lucas, *Arch. Int. Med.*, 1906, xvii., 543.

Hæmorrhages in New-born Children.

Cases of hæmorrhage in infants at, or soon after birth, may be divided into three groups :

1. **Traumatic Cases.**—The experiences of special strain and stress which many infants come through during their passage into extra-uterine life often result in internal hæmorrhages of various degrees of severity. These may occur in the brain or meninges, in the abdominal or thoracic organs, or in the muscles and superficial tissues ; and the extreme congestion of the circulation, as well as the strain the muscles are exposed to, goes far to account for the tendency there is to their occurrence.

To prevent the difficult labours that cause them, the mothers should always be examined periodically during their pregnancy and everything possible done to rectify breech-presentations and other wrong positions before it is too late to do so. It is also advisable, if the infant seems much congested at the time of birth, to allow a teaspoonful or two of blood to escape from the cut end of the cord to relieve his circulation. It is well also to avoid too vigorous attempts to establish respiration—*e.g.*, in using the Schulze method—as this might start or increase hæmorrhages into the congested tissues. In cases in which bulging of the fontanelle indicates the presence of intracranial hæmorrhage, repeated lumbar puncture may also be done with advantage.

2. **Symptomatic Cases**, in which purpura occurs during the early months of life as a symptom of some disease such as congenital obliteration of the bile-ducts, septicæmia, syphilis, or congenital heart disease. In these the inclination to bleed continues indefinitely along with the original malady.

3. **The Hæmorrhagic Disease of New-born Children.**—This disease of unknown origin occurs in apparently healthy infants. It attacks boys and girls equally. It is quite definitely not a manifestation of hæmophilia, it is not due to sepsis, and neonatal injury plays no part in its causation. Hæmorrhage may occur in congenital syphilis, but lues certainly is not an etiological factor in the vast majority of the cases.¹

Normally the blood coagulation time in infancy lengthens during the second, third, and fourth days, which is the time of greatest incidence of the disease, and Capon² has suggested

¹ R. S. Beveridge, *Arch. Dis. Child.*, 1928, iii., 39.

² N. B. Capon, *Lancet*, 1924, i., 1203.

that the pathogenesis in part at least may be dependent on a delay in reaching the normal level.

The hæmorrhage may occur from many sites—the umbilicus, the stomach and intestines (*melæna neonatorum*), the vagina, the nose, or the skin; and in fatal cases hæmorrhage into the brain, meninges, lungs, pleura, pericardium, and abdomen has been observed.

The bleeding varies in severity; in some cases it is slight and easily controlled and causes the child little disturbance, but in other cases it is severe, and comparatively soon the child appears as if exsanguinated. As previously mentioned, the most common time for the hæmorrhage to occur is on the second, third, or fourth days, but it may appear immediately after birth or be delayed for as long as the fourteenth day.

The *diagnosis* is usually quite apparent, but care must be taken not to mistake bleeding from an injury of the mouth or melæna consequent on the child swallowing blood from epistaxis or from the cracks in the mother's nipple.

Treatment.—One very important point about this condition is that, whatever the cause may be, it acts only for a short time, so that prompt and energetic treatment is of the utmost importance.

A subcutaneous injection of 10 c.c. of whole blood from either of the parents, or the nurse or friend, if carried out at once, almost invariably brings about a speedy cure. If this treatment has been delayed and the child is suffering from loss of blood, or if the subcutaneous injection of blood has not been successful, then a blood transfusion is called for. The mortality of cases treated by transfusion is almost nil. For the technique of blood transfusion see Chapter XXXIX, p. 1014. Horse serum or any available serum may be used instead of blood and has often proved quite satisfactory, but as the essence of the treatment is that it should be carried out early there should be little call for this method, as human blood will always be more readily obtained.

In cases of *melæna neonatorum* the child should be kept as quiet as possible, wrapped in cotton wool, and surrounded by hot-water bottles. If very weak, he should not be allowed to suck, but should be fed with a spoon, or syringe and tube, with small quantities of breast-milk or peptonised milk cooled on ice. Injections into the bowel are inadmissible, because they do harm by stimulating the intestinal movements.

CHAPTER XXI

DISEASES OF THE DUCTLESS GLANDS

The Thymus

THE authorities who have written on the thymus have differed greatly as to what is to be considered its normal weight. It is certain that the older writers gave far too high a figure, owing to their taking as their standard the thymus glands of infants who had died suddenly during apparently normal health. We now know that it is just in such children that we may expect to find an enlargement of the organ. Thus Friedleben¹ gives 20 grammes as the normal weight; while more recent writers, such as Bovaird,² Thursfield,³ and Dudgeon,⁴ place it at 6 or 7 grammes only, and regard anything over 10 grammes as abnormal. The recent findings of the Status Lymphaticus Investigation Committee of the Medical Research Council⁵ are, however, more in agreement with those of Friedleben. According to this investigation the average weight of the thymus varies between 25 grammes at birth and 34 grammes at sixteen years, and the gland is slightly heavier in males than females.

The gland was formerly supposed to increase in size during the second year; but the above-mentioned Committee found that it changed little till after six years of age. Its bulk varies greatly in different individuals, and also in the same individual at different times, according to the state of his nourishment. There is no other organ in the body which changes so much in size; and it is very difficult, if not impossible, to make sure by percussion during life how far it really is enlarged; even an X-ray examination often gives inconclusive results.

¹ *Die Physiologie der Thymusdrüse in Gesundheit und Krankheit*, Frankfurt a. M., 1858.

² *Arch. of Pediat.*, Sept. 1906, 641.

³ *St Bart. Hosp. Rep.*, 1903, xxxviii., 129.

⁴ *Trans. Path. Soc. Lond.*, 1904, lv., ii., 151.

⁵ *Journ. Path. and Bact.*, 1931, xxxiv., 213.

The functions of the thymus are still very obscure. Park and McClure,¹ as also Renton and Robertson,² have found that, in the dog, it is not essential to life, and that its removal produces no detectable alteration in the hair, teeth, contour of the body, muscular development, strength, activity, or intelligence of experimental animals, and probably has no effect on growth or development, although it may possibly cause retardation in development and delayed union of the epiphyses. They also find that extirpation of the thymus produces no lasting changes in other endocrine organs, although possibly some alterations in these may occur during the period immediately following its removal.

For such a large and characteristically infantile organ the thymus has strangely little clinical significance so far as we know. Thymus atrophy, however, is the most characteristic change found in the bodies of atrophied babies—apart from the wasting of their fat and muscles. It is even said that the state of nutrition of an infant may be estimated by a microscopical examination of its thymus.³

Status Lymphaticus (*Lymphatism*).

The status lymphaticus is a condition of bodily debility characterised by hyperplasia of the thymus and of the lymphoid tissue generally throughout the body, especially that at the root of the tongue, in the mucous membrane of the bowel, and also to a less obvious degree in the thyroid. This is accompanied by a great lowering of the patient's power of resistance, and is believed to account for a large number of otherwise unexplained cases of sudden death.⁴ So far as death from operation or anæsthetic shock is concerned, the M.R.C. Committee found no evidence of enlargement of the thymus in their series of cases.

Various suggestions have been made as to why such a lymphatic habit of body should give rise to so dangerous a state of weakness of the heart and nervous system, and the question is still undecided. The old view, that the symptoms

¹ Edwards A. Park and Roy D. McClure, *Amer. Journ. Dis. Child.*, Nov. 1919, 479.

² J. M. Renton and M. E. Robertson, *Journ. Path. and Bact.*, 1916, xxi., 1.

³ Stokes, Ruhräh, and Rohrer, *Amer. Journ. Med. Sci.*, Nov. 1902, 847.

⁴ C. McNeil, *Edin. Med. Journ.*, Jan. 1914, 25.

are due to a sudden congestion of the enlarged thymus causing dangerous pressure on neighbouring vital parts, is now generally abandoned in favour of Paltauf's hypothesis¹ of a morbid diathesis. Escherich² believed that a sort of hyperthymisation of the blood takes place, which keeps up a constant state of irritable weakness in the nervous system, so that trivial causes have unexpectedly severe effects. He regarded lymphatism, therefore, as due to a disordered action of the thymus, somewhat in the same way as myxœdema and exophthalmic goitre depend on disordered thyroid action.

Symptoms.—The so-called lymphatic child is unduly obese, flabby, and pale. Many of them would pass for prize babies. During infancy rickets and spasmophilia are not uncommon. The tonsils and adenoid tissue in the nasopharynx, and at the root of the tongue, may be hypertrophied and the spleen palpable. Some enlargement of the lymphatic glands is often found and a lymphocythæmia is also described.

Occasionally the state of the thymus may be recognised from the amount of dullness on percussion found over and near the manubrium sterni. Within recent years it has become popular to decide whether the thymus is enlarged or not from appearances in the radiogram. But everyone with any experience of radiology for this purpose must have become rather sceptical because of the variations that may be present from day to day in the size of the supra-cardiac mediastinal shadow—the so-called thymus shadow. We know that the size of this shadow is influenced by respiration, being wider during expiration from shortening of the mediastinum and narrower during inspiration through the fall of the diaphragm and lengthening of the mediastinum. Hasley and De Tomasi³ have further shown from cinematographic studies that the size of the shadow is also influenced by the state of the heart and large vessels. During cardiac diastole and engorgement of the great vessels the shadow becomes increased and at times is so marked as to suggest enlargement of the gland. These authors conclude that hypertrophy of the thymus is much less common than is usually supposed.

¹ *Wien. klin. Wochenschr.*, 1889, ii., 877 ; and 1890, iii., 172.

² *Berlin. klin. Wochenschr.*, 1896, xxxiii., 645.

³ C. K. Hasley and R. Q. De Tomasi, *Journ. Mich. State Med. Soc.*, Jan. 1930.

Generally, however, the presence of lymphatism is not suspected unless serious symptoms set in. These may consist in a succession of ill-defined convulsive seizures, accompanied by faintness, cyanosis, and dyspnœa, and they may recur at intervals for weeks or months. Stridulous breathing (*thymic asthma* or *stridor*) in the young infant is considered by some authorities a frequent manifestation of an enlarged thymus. When the thymus is the cause it is said that the stridor is present both during inspiration and expiration, and that it is influenced by the position of the head. When the head is extended, owing to the isthmus of the thorax between the sternum and vertebral column becoming narrowed, the symptoms become increased in severity, while flexion of the neck acts in a converse fashion and relieves the stridor. Hence it is recommended that infants with this complaint should be made to sleep with the head resting on a high pillow. In our experience these tests, as well as the radiological evidence, have been so conflicting that no definite conclusions regarding the state of the thymus could be drawn. It seemed more likely that the condition was of the nature of congenital laryngeal stridor (p. 655).

In most fatal cases the syncope follows some trivial exertion or shock, without any previous warning of danger. The child, who has been regarded as in good health, or, perhaps, as only flabby and unenergetic, suddenly becomes faint, gasping, and cyanosed, his eyes turn up, and he loses consciousness. Convulsive movements may or may not occur. The heart stops before the respiration; a fatty condition of the myocardium is usually found after death.

There is sometimes no apparent exciting cause for the seizure, but generally some shock of a trifling kind has preceded it. A sudden plunge into water, a wet pack, a hypodermic injection, even the application of a tongue depressor, have been described. Not infrequently the child is found dead in his bed or perambulator. Some sudden deaths under anæsthesia or during convalescence from such diseases as diphtheria and typhoid are supposed to be due to this condition.

McNeil and McGowan¹ have suggested that certain malignant epidemics of pneumonia, and also of scarlet fever, may be

¹ *Edin. Med. Journ.*, Mar. 1913, 201; and C. McNeil, *Edin. Med. Journ.*, Jan. 1914, 25.

due to a complication of lymphatism with the other diseases. There would seem no doubt that this type of child, whether the thymus is an active participant or not, is specially susceptible to infection, to which he reacts unduly.

The **diagnosis** of lymphatism must often remain merely a matter of conjecture. When its presence is suspected, the **prognosis** must always be extremely guarded, for any child whose thymus is greatly enlarged is likely to have a very slender hold on life; nevertheless, it is probable that many children outgrow the condition.

Treatment.—If the status lymphaticus is diagnosed or suspected, we must be exceedingly careful about administering an anæsthetic, or even a wet pack, or doing the smallest operation that is not urgently called for. When syncope actually sets in, no known treatment is of any avail.

A number of more or less active forms of treatment have been suggested to reduce the hypertrophy. Excision of as much as possible of the organ has been recommended by many, but the operation is not without danger, and the results have been discouraging. Friedlander¹ and Ratchford² have reported improvement from the use of X-rays.

Diseases of the Lymphatic Glands.

It is doubtful whether the lymphatic glands produce a real internal secretion, but it is convenient to consider them here along with the other ductless glands. Their peculiar characters in children have already been dealt with (p. 76); and their tuberculous affections are considered in Chap. XXXVII, p. 960.

Lymphadenoma (*Hodgkin's Disease*).—Lymphadenoma is not a common disease in children. It usually occurs about the time of the second dentition, and its symptoms do not differ from those in later life. In the earlier stages the general health is little affected, and the chief symptom is great enlargement of the cervical glands, which, unlike those in tuberculous conditions, are soft, discrete, movable, and painless. There may also be enlargement of the spleen, which usually has a somewhat irregular surface, and of the liver. Later, the glands in the axilla, groin, and elsewhere also increase in size. In

¹ *Arch. of Pediat.*, 1911, xxviii., 810.

² *Amer. Journ. of Med. Sci.*, 1910, cxi., 550.

time the enlarged glands become matted, and pressure symptoms occur from involvement of those in the thorax and abdomen. Other symptoms are distension of superficial veins, œdema of the lower limbs, serous effusions, respiratory embarrassment, jaundice, and general exhaustion with failure of circulation. Hæmorrhages rarely occur, and there is nothing characteristic to be found on examination of the blood, though at some period there is a leucocytosis.¹ Short periods of pyrexia—continuous, intermittent, or remittent—may alternate with periods of normal temperature (Pel-Ebstein type). The cause of the disease is quite unknown.

The *diagnosis* is difficult and as a rule can only be arrived at from the histological examination of an excised gland. In the majority of suspected cases tuberculosis is the cause. In lymphosarcoma the disease runs a more acute course and jaundice is an earlier symptom; the spleen is usually normal in size, the enlargement of the liver is greater, and there is not the same general glandular involvement. The *prognosis* is altogether bad, probably no genuine case of the disease recovers. The end usually comes within two or three years. Occasionally rapidly fatal cases are met with, and a few live for many years.

Treatment is of little use. Temporary improvement sometimes follows the use of arsenic. With X-rays the glands may entirely disappear, but they usually recur.

Lymphosarcoma.—Lymphosarcoma is commoner in children than in adults, though it is not a disease of early infancy. The disease generally begins in the posterior mediastinal glands, but its first clinical manifestation is usually a moderate enlargement of the cervical glands; in a few cases the cervical disease is the primary lesion. For some time after the glands enlarge the general health remains good. At first the glands are soft, movable, painless, and not adherent to the skin or deeper tissues; but these become involved later. There is no characteristic change in the blood. As the disease progresses, other symptoms develop, such as loss of appetite, anæmia, breathlessness, choking fits, giddiness and faintness, slight cough, and general debility. These go on increasing, diarrhœa and vomiting may occur, the spleen ultimately enlarges and ascites sets in with œdema of the lower limbs; and the child gradually sinks and dies.

¹ R. Muir, *Text-book of Path.*, London, 1924, 399.

The *diagnosis* is often difficult at first. The glandular swelling differs from that in tuberculous adenitis in showing no signs of periadenitis and being soft throughout, painless, and non-adherent until the later stages. The general health is less affected, and there are usually more signs of mediastinal involvement. In lymphadenoma the glands remain discrete much longer, while in lymphosarcoma there is less tendency to general enlargement of glands and of the spleen.

When the disease has begun in the mediastinal glands, the condition is hopeless; but, if there is no sign of involvement of the thoracic glands, it is certainly well to have those in the neck excised, in the hope that the case may be one of the rare instances of primary gland affection. In one case (a boy of four years and nine months), reported by D. M. Greig,¹ the child was in perfect health eleven and a half years after the operation.

The Thyroid and its Diseases.²

During recent years a great deal of work has been done on the physiology and pathology of the thyroid gland, and its wide-reaching influence on the general health of the body at all periods of life is now recognised. The small size of the gland, its isolated position, and the unobtrusive nature of its functions have combined to render the clinical study of its diseases specially difficult, so that as yet only a few of the most serious of them can be recognised during life. All modern authorities seem to be agreed that the efficiency of the infant's thyroid depends largely on the healthy action of the mother's gland during his intra-uterine life. If this is impaired, his thyroid tissue is apt to suffer; and it may undergo hypertrophy, cellular necrosis, or fibroid change.

During the early months of lactation, the mother's thyroid increases its activity in order to supply the wants of the infant through her milk and supplement the scanty secretion which his own gland provides. As the calf's thyroid is earlier than the child's in developing fully, cow's milk may not supply the baby with as much thyroid secretion as he receives from his mother's milk. In the later months of lactation, and especially

¹ *Brit. Journ. of Child. Dis.*, Nov. 1907, 469.

² R. McCarrison, *The Thyroid Gland*, London, 1917.

after weaning, the infant's thyroid secretion becomes more active, and it remains so during childhood.

The **causes** of abnormal thyroid action (apart from neoplasms) may be either nutritional, toxic, or psychical.

Among the *nutritional* causes are (a) a defective or improper food supply (too little food or food which contains too little iodine), (b) too much food or too much butcher-meat, and (c) insanitary surroundings (bad air or an impure water-supply).

Bacterial and other toxæmias and infectious maladies of many kinds may have an unfavourable effect on the action of the thyroid, as also may rickets, lymphatism, and other debilitating diseases; but it is extremely rare to have thyroiditis set up by any of these conditions. A very important cause of thyroid disease is the occurrence of toxæmia arising from the stomach, the bowels, or the teeth.¹ This poisoning may cause hypertrophy of the gland followed by atrophy, or primary atrophy may occur without previous enlargement.

Like the suprarenals, the thyroid is capable of being incited to excessive action by *psychic influences*, such as fear and other strong emotions.

Thyroiditis.—Acute inflammation of the thyroid, which can be clinically diagnosed as such, is a very rare disease; but acute and chronic forms of thyroiditis which cause no symptoms are probably less uncommon. They occur as complications of acute infectious diseases, or from intestinal and other toxæmias, and are only recognised afterwards by the resulting thyroid deficiency. The chance of the thyroid recovering its function probably depends largely on the state of the child's general nutrition at the time of his convalescence.

Acute thyroiditis is occasionally simulated by septic inflammation of the adjacent lymphatic glands. When the organ itself is inflamed, the resulting swelling is painful, and moves with swallowing; and the child holds the head forward with the neck stretched out. There may also be a degree of dysphagia and dyspnœa with a peculiar stridor. An abscess generally forms.

Enlargement of the Thyroid.—A slight degree of thyroid swelling is common temporarily in girls about the time of puberty. It is sometimes associated with delayed or irregular menstruation, with chronic intestinal disturbance, and functional nervous symptoms. It usually has no serious significance.

¹ Osborne, *New York Med. Journ.*, 2nd Mar. 1918, cvii., No. 9, 385.

Chronic enlargement of the thyroid (simple, cystic, and adenomatous goitre) may occur sporadically, or as an epidemic; and, although endemic cretinism has for a long time practically ceased to exist in this country, endemic goitre is still prevalent in a mild form, in various places such as the Thames Valley, the Dales, Derbyshire, Sussex, Hampshire, and Lanarkshire. The condition is usually very chronic and causes little inconvenience. Its etiology is probably somewhat complicated¹; but far the most important causes from a practical point of view are (a) a deficiency of iodine, actual or relative, in the food, or a failure on the part of the body to assimilate what is present; and (b) "an organism or organisms present in the gastro-intestinal tract and introduced into it by polluted water or food."

The *prevention and cure* of goitre depend on two main factors: (1) the amount of iodine in the diet available for the needs of the thyroid gland and those of the body generally; and (2) the general hygiene of the individual, especially that of the intestinal tract. In some cases the first of these may be the more important, and in others the second; but practically it is always well that the bowels should be carefully attended to, a pure water-supply secured, and small doses of iodide taken regularly. For treatment, two or three grains of iodide thrice daily will probably suffice. For prevention, Marine and his co-workers² recommend "2 grams of sod. iod. in 0.2 gram doses distributed over a period of two weeks, and repeated each spring and autumn." These doses have been found to cause no inconvenience to the children taking them, and to be extremely successful in preventing the occurrence of the disease. Care must, however, be exercised as this treatment sometimes converts a simple into a toxic goitre.

Gummata and *tuberculous nodules* are occasionally found, post-mortem, in the thyroid, but they are not of clinical importance.

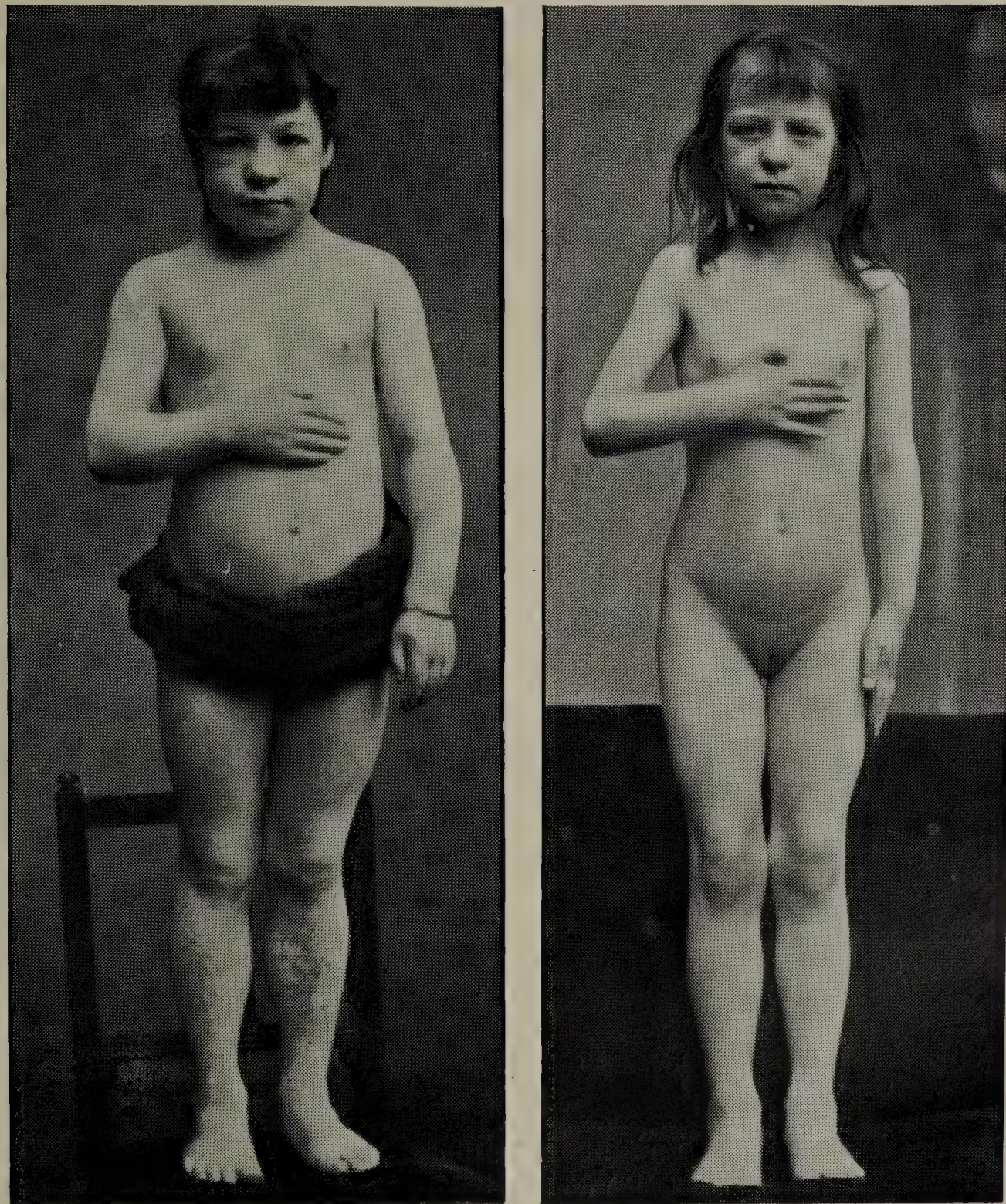
Graves' Disease.—Exophthalmic goitre is very rare indeed in early childhood, and is uncommon in a typical form in older children. The worst case we have seen was in a boy of ten, in whom the symptoms set in immediately after a Zeppelin raid. Sometimes, in cases of chorea, we find mild

¹ Rob. McCarrison, *Brit. Med. Journ.*, 1924, i., 989.

² David Marine, C. H. Lenhart, O. P. Kemball, and J. M. Rogoff, *Western Reserve Univ. Bull.*, Cleveland, July 1923, xxvi., 1-123.

symptoms of Graves' disease—thyroid enlargement, slight exophthalmos, von Graefe's sign, and a quick pulse.

The prognosis of Graves' disease in children is always good, although the recovery may be slow. Nearly all the cases we have



FIGS. 146 and 147.—Juvenile Myxœdema, before and after thyroid treatment. (Girl of 12 years.)

seen have been in hospital practice; and the rest and quiet in the ward, with careful dieting, have in every instance led to improvement in the general health, and to steady recovery from the special symptoms. Surgical treatment is never necessary in childhood.

Myxœdema.

It is often impossible to distinguish myxœdema, which has originated in infancy, from congenital cretinism; and, practically, it is not important to do so. In rare instances, however, we meet with typical cases of the disease beginning in older children which do not differ in symptoms or treatment from those in adults (Figs. 146 and 147). One of us (L. F.) has seen myxœdema in a mother and child.

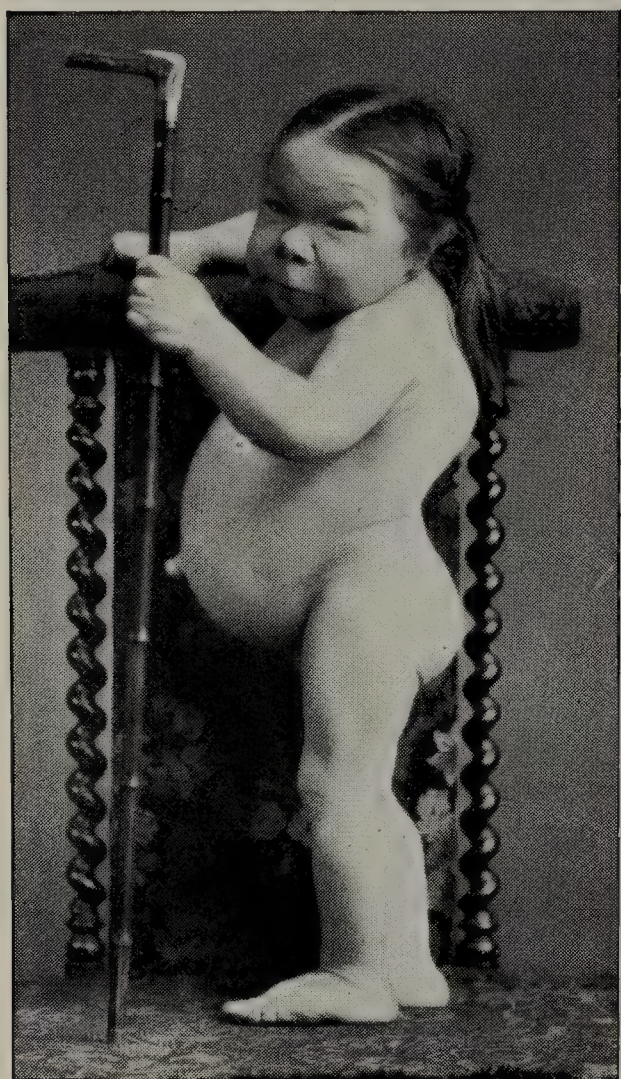


FIG. 148.—Sporadic Cretinism, untreated.
(Girl of 22 years.)

Endemic cretinism has almost ceased to occur in Great Britain. Nevertheless, most of the cases observed at the Royal Hospital for Sick Children, Glasgow, were resident in the Wishaw district of Lanarkshire.

Sporadic Cretinism.

Causation. — The primary cause of the destruction of the thyroid which produces cretinism is still entirely unknown. The disease generally appears in quite healthy families, though occasionally more than one child is affected.

Presumably the disease may be due to a poison, produced in the mother's intestine during pregnancy, acting on the thyroid of the foetus; but, on careful

inquiry into the mother's health during pregnancy in more than fifty cases, one of us (J. T.) was scarcely ever able to find evidence of any ill-health on her part to which the condition of the foetus could be ascribed. In four cases the mothers subsequently developed a small goitre, but in none of them did recognisable symptoms of myxœdema occur. Another mother, who had two cretins in her family, developed slight myxœdema twelve years after the first of them was born. The fact, however, that most of the cases in the West of Scotland emanated from one

locality suggests a cause other than that of some toxin of parental origin.

Symptoms.—The characteristic appearances of cretinism as it is seen in adults and older children are readily recognised. The extremely stunted growth, large head, relatively short, thick-set limbs, thick, dry redundant skin with supraclavicular swellings, bloated features, subnormal temperature, and other well-known symptoms, form a clinical picture which is not easily forgotten or mistaken for anything else (Figs. 148 to 150).

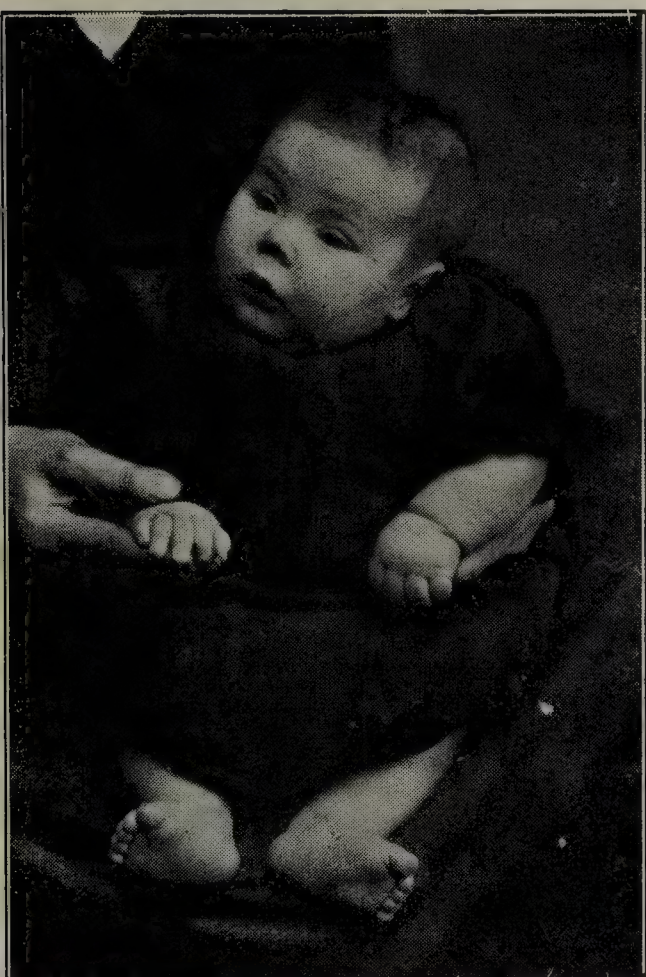


FIG. 149.—Sporadic Cretinism. (Boy of 7½ months.)



FIG. 150.—Sporadic Cretinism. (Girl of 15 months.)

Usually the condition is less easy to recognise in infants than in older patients.

In the great majority of cases, however, a confident diagnosis may be made very early, if (1) we obtain a careful history from the mother of the child's bodily and mental development, of his demeanour, and of the characters of any illnesses he has had; and (2) if we examine him thoroughly for evidence of delayed development and for signs of myxœdematous infiltration of any of the subcutaneous, submucous, or other tissues.

(1) It will be found that the child has generally been *backward or peculiar* in some way or other in his conduct. He

has often from birth been chilly, apathetic, and behind other infants of his mother's acquaintance in showing interest in what he sees and hears. His speech has been long in coming and very scanty, and he has been late in learning to use his hands, and in sitting up, standing, and walking. In severe cases there may have been difficulty in getting him to take the breast or bottle, so that he has had to be fed with a spoon. Often we are told that he has scarcely ever laughed; and that, though he has whimpered a good deal at times, he has rarely, if ever, cried right out in a hearty infantile way.

In a considerable proportion of the cases the child will have been noticed, during the early months, to have had a habit of keeping his tongue protruded between his lips; and usually he has been much given to snoring and sneezing. In most instances, also, there has been more or less constipation; and the teeth have been long in appearing. Very late teething, however, is not so common as is stated in many of the text-books, for in 37 cases in which this point was ascertained, the first teeth had appeared by the ninth month in 16, and about the end of the first year in 28, and only in one as late as two years. Delayed dentition is much commoner and more characteristic in mongolism.

When we inquire about *previous illnesses and symptoms*, we usually find that the child has shown none of the tendency to recurrent catarrh of the different mucous membranes which is so common in mongols; but such ailments as he has had may have shown peculiar features. If, for example, he has had any inflammatory or other feverish illness—such as an abscess, pneumonia, or influenza—his temperature will have been little if at all raised above the normal; and the usual nervous disturbances which accompany such complaints will have been slight or absent. Thus, we have had two cases in which cretin babies were found to have died from pneumococcal meningitis—the whole vertex being covered with purulent lymph—although they had shown scarcely any rise of temperature, no vomiting, and no other distinctively cerebral symptoms apart from drowsiness. Recurrent seborrhæic dermatitis, on the scalp and elsewhere, is fairly common in infancy; and, during early childhood, tuberculous lesions of the bones and of the mesenteric and other glands occur not uncommonly. We are also often told that the child has had chronic bronchitis.

His peculiar behaviour and demeanour are often significant. In the early months he lies unnaturally quiet in his cot and makes few of the instinctive aimless movements that we look for in a normal baby of his age. Later, too, he shows far too little spontaneous restlessness, and his actions are like those of a much younger infant. As he grows older there is a curious air of apathy about him and an abnormal deliberation in all that he does. He seems also to have too little initiative, and shows little inclination to do many of the little things that ordinary babies take pleasure in. He knows, however, the few things he does wish to do, and quietly insists on doing them—though he is very long about it. Even if not at all imbecile, the cretin baby is apt to appear so, because he is so unresponsive in his demeanour. He is, however, really quite observant in his own quiet way, and takes in a great deal more of what is going on than we may at first sight give him credit for. His eyes lack the bright alertness of early childhood, and their expression is serious and nearly always sad, but they have a steady, patient look which is very different from the shifting, vacant expression seen in most imbeciles.

(2) On *examining the baby* we generally find quite enough in his appearance and physical signs to establish the diagnosis—provided, of course, that he has not been given thyroid gland before we see him—for even a week or two of treatment usually removes many of the characteristic features of the disease. On palpation, it is generally easy to recognise that the thyroid is absent or much too small. We also find, in many of the older children, that the skin and hair feel dry and harsh, although this is rarely so in infants. The peculiar brick-red circumscribed flush on the cheeks which is common in adult myxœdema is only rarely seen in young children (Fig. 151).

The body and limbs always show some degree of stiffness; but in a few cases the general muscular rigidity is so great that the condition resembles spastic diplegia. We have seen three instances of this among 62 cretins. This type of case seems to correspond to that which McCarrison found among endemic cretins in India, and has described as “nervous cretinism” (Fig. 154, p. 511). The rigidity passes off under thyroid treatment. It has been suggested that this peculiar type of case is connected with involvement of the parathyroids, but if this be so it is difficult to understand the effect of the administration of thyroid extract.

In all the cases we have seen the mental development was of a low grade and did not improve much under treatment, although the bodily growth progressed in the usual way. X-ray examination of the wrists will invariably show delayed ossification (Figs. 152 and 153).

The cretin's temperature is always subnormal. He also shows various indications of retarded bodily development, some of which have been already mentioned. The fontanelle is large, and usually remains widely open in later childhood and adolescence. We have found it so in an untreated cretin of



FIG. 151.—Sporadic Cretinism. (Girl of $2\frac{1}{2}$ years, showing circumscribed flush on the cheeks.)

thirty-six years. The cranium is of normal dimensions and well ossified apart from the state of the fontanelle. Although the child is usually normal in size and proportions at birth, his gain in length as he grows older is subnormal, so that older cretins are always more or less dwarfed in stature.

In addition to evidence of delayed development, we generally find physical signs and symptoms which indicate the presence of *myxædematous thickening of the subcutaneous, submucous, and other tissues*; and the rapidity with which these disappear when thyroid is given shows that their cause must have been of this nature. Although during early infancy we rarely find much myxædematous swelling of the superficial subcutaneous tissues

or in the muscles, and never very prominent supraclavicular or pre-axillary pads, there is usually enough in various parts of the body to be distinctly recognisable. Nearly always we find some puffiness of the features, with thickening of the eyelids, nostrils, and lips, also of the hands and feet; and the back of the neck is much thickened (Fig. 159, p. 515). The infiltration of the scalp tissues often leads to an exaggerated furrowing of the forehead, especially when the baby cries.

The swelling of the submucous tissues in the nose and nasopharynx is shown by the child's snorting and snoring breathing;



FIG. 152.—Skiagram of hand of Untreated Cretin aged 5 years.



FIG. 153.—Skiagram of hand of Normal Child aged 5 years.

and in many cases the changes in the larynx give to the cry a peculiar harsh, leathery, toneless quality which is most characteristic. Frequently also there are loud rhonchi all over the chest, which seem to be due to similar changes in the lining membrane of the bronchi.

The enlargement of the tongue, which leads to its habitual protrusion in about half the cases in infancy, is also the result of myxœdematous infiltration. Tongue sucking, followed by enlargement of the papillæ and fissuring of the mucous membrane, is not very uncommon. Some considerable degree of abdominal fullness is always present, and an umbilical hernia is found in more than four-fifths of the cases.

As the child grows older his development lags behind, and the characteristic cretinous appearance becomes steadily more striking. The myxœdematous swelling increases in the face and elsewhere, the belly becomes more prominent, and marked lordosis develops. Circumscribed soft swellings form above the clavicles and in front of the axillæ. The dryness of the skin increases, and in later childhood the hair also becomes scanty and dry. The fontanelle remains widely open. The milk-teeth generally remain far too long in the gum, and they may all be present as late as the nineteenth year. The child's growth and activity are greatly interfered with, so that at ten or twelve years old he often has not the size or strength of a boy of three or four. The mental condition in an ordinarily severe case is that of imbecility, but in slighter forms of the disease the child seems merely backward. He is dull and apathetic, slow of movement and of apprehension, but neat and tidy in his ways, and quiet and docile unless he is teased. Speech is generally long in being acquired, and the words used are few.

Diagnosis.—Slight cases of cretinism are sometimes met with (Fig. 155) in which there is little beyond stunted growth, delayed closure of the fontanelle, and some degree of mental dullness. Confirmation of the diagnosis is afforded by the remarkable improvement which follows thyroid treatment.

The two morbid conditions most apt to be mistaken for cretinism are mongolism and achondroplasia. In mongolism the likeness is not very great even in older children. In infancy the mongol and the cretin have really little in common

	Cretin.	Mongol.
Complexion	Sallow	Ruddy
Skin	Tends to dryness	Soft and natural
Neck	Thick	Slender
Thyroid	Not felt	Felt
Circumference of cranium .	Normal or large	Usually subnormal
Obliquity of palpebral fissures	Absent	Generally present
Hyperextensibility of joints .	Absent	Present
General stiffness of body .	Present	Absent
Demeanour	Stolid and apathetic	Lively and inquisitive
Recurrent catarrh of mucous membranes	Rare	Very common
Congenital defects and malformations	Absent	Often present
Temperature	Subnormal	Normal

beyond the mental backwardness, the frequently protruded tongue, and the general fact that they are rather ugly babies. In cretinism the features are merely coarsened, whereas in mongolism the features are really deformed.

Some of the principal differences between cretins and mongols may be tabulated as shown on opposite page.



FIG. 154.—Sporadic Cretinism. Nervous type. (Girl of 7 years.)



FIG. 155.—Sporadic Cretinism. Slight case. (Girl of 5 years.)

The resemblance which achondroplastic dwarfs have to cretins at birth is in some ways striking, but it is *adult* cretins that they resemble (Fig. 272, p. 870).

Treatment.—This consists in the continued administration of some preparation of thyroid by the mouth. The fresh raw gland is probably more active and trustworthy than any of its preparations; but nowadays it is rarely used. It may be given to a young child in doses of $\frac{1}{16}$ to $\frac{1}{8}$ of a lobe *twice a*

week to begin with, and may be increased according to its effect. It is a curious fact that the improvement in cases where thyroid is given twice a week seems usually just as continuous and satisfactory as that seen when the remedy is administered in small doses daily or every few hours.

Generally, however, it is much more convenient to use one of the manufactured preparations and to give it once daily. The tabloids prepared by Messrs Burroughs & Wellcome are the form most widely used, and they do well, provided they are kept dry. A tabloid of 1 to $2\frac{1}{2}$ gr. of gland substance may be given to a young infant to begin with. If no unpleasant symptoms arise, the dose may be repeated on the third day, then given every second, and later every day. In older children twice these doses or more may be used.

The effect of the first few doses should be carefully watched, because in rare cases thyroid, even in very small doses, causes serious symptoms of poisoning. We have seen two such cases among 62 cretins of various ages. In one of these, a boy of 19 months, acute diarrhoea followed even a single grain, so that, after repeated trials, the treatment had to be given up. The child died shortly after from measles and broncho-pneumonia. The other case was that of a girl of 7 years, with spastic symptoms (Fig. 154), in whom thyroid caused alarming attacks of faintness and vomiting. These so frightened her mother that she refused to continue the treatment. Some years later she was taken into the Larbert Institution where, under cautious administration of small doses, she gradually lost her special susceptibility to the drug, and did well bodily, although she remained a low-grade imbecile.

In a large proportion—more than a half—of the young children with sporadic cretinism, thyroid treatment, while agreeing well in other ways, sets up obstinate enuresis. This does not, in our experience, yield to belladonna or any other drug; and in some cases it makes it necessary to lessen the dose and retard the child's progress towards recovery. In time, however, all the cases recover.

The right dose of thyroid differs considerably in different cases and must be determined in each by watching the effect of the remedy on the child's appearance, vigour, weight, growth, and temperature. If he is gaining satisfactorily in height and is fairly active, and if his general condition is good, the dose

is probably sufficient. If he is not growing as he should in height, is gaining weight steadily, and is remaining torpid and disinclined for exertion, it will be well to increase the amount of thyroid given. If, on the other hand, although growing in length he is losing weight continuously, sweating much, and showing signs of nervousness, it should be lessened.

During treatment, except in severe or debilitated cases, the child may go about as usual, and no special diet is required. Should he become feverish and develop sickness, headache, and malaise, this shows that much too large a dose has been given. He should under these circumstances be kept in bed and the thyroid stopped for some days.

When thyroid treatment is begun for the first time at puberty or during adolescence, it is important to keep the patient off his feet for a large part of the day. At these ages the thyroid causes softening of the shafts of the long bones, and unless great care is taken to prevent it, severe bowing of the legs will develop.¹ This danger does not seem to exist in young children.

Result of Treatment.—When the thyroid is carefully given, rapid and continuous improvement practically always follows (see Figs. 156 to 165). The temperature rises to, and remains at, the normal level. The unnatural swelling quickly disappears from the face and other parts of the body. The features become more sharply defined and more mobile and expressive, and the eyes brighten. At the same time the tongue ceases to be protruded, the voice becomes less guttural, the wheezing stops, and the child no longer snores at night. The abdomen diminishes rapidly in circumference; and, if a small umbilical hernia has been present, it disappears. The supraclavicular swellings vanish at an early stage of the treatment. The skin loses its harsh and dry feeling and becomes soft, and the cheeks show a natural flush. In young patients the hair sometimes falls out, at first, in considerable quantities; but it is soon replaced by a new crop, which grows more rapidly and is softer and often of a different shade of colour. The joints are no longer kept slightly flexed, the limbs become firm and strong, and the back also grows straighter and more shapely. The retarded evolution of the teeth is actively resumed. If thyroid

¹ "Variations in and Limits of Improvement of Cretins at Different Ages under Thyroid Treatment," *Brit. Med. Journ.*, 1896, ii., 618.

THYROID TREATMENT OF CRETINISM.



FIG. 156.—Sporadic Cretinism. (Girl of 17 months.) Before treatment.

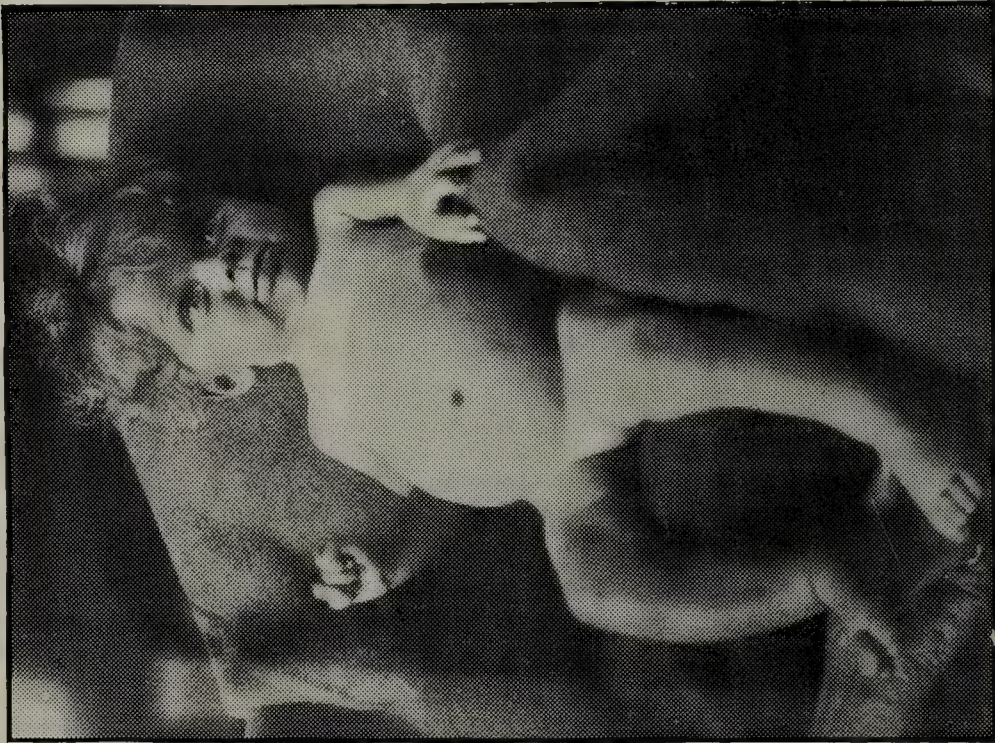


FIG. 157.—Sporadic Cretinism. Same patient after 1 month's treatment.



FIG. 158.—Sporadic Cretinism. Same patient after 3 months' treatment.



FIG. 159.—Sporadic Cretinism. Back view of same before treatment.



FIG. 160.—Sporadic Cretinism. Same patient after 3 months' treatment. Back view.



FIG. 161.—Sporadic Cretinism. Same patient after 12 months' treatment.

THYROID TREATMENT OF CRETINISM.

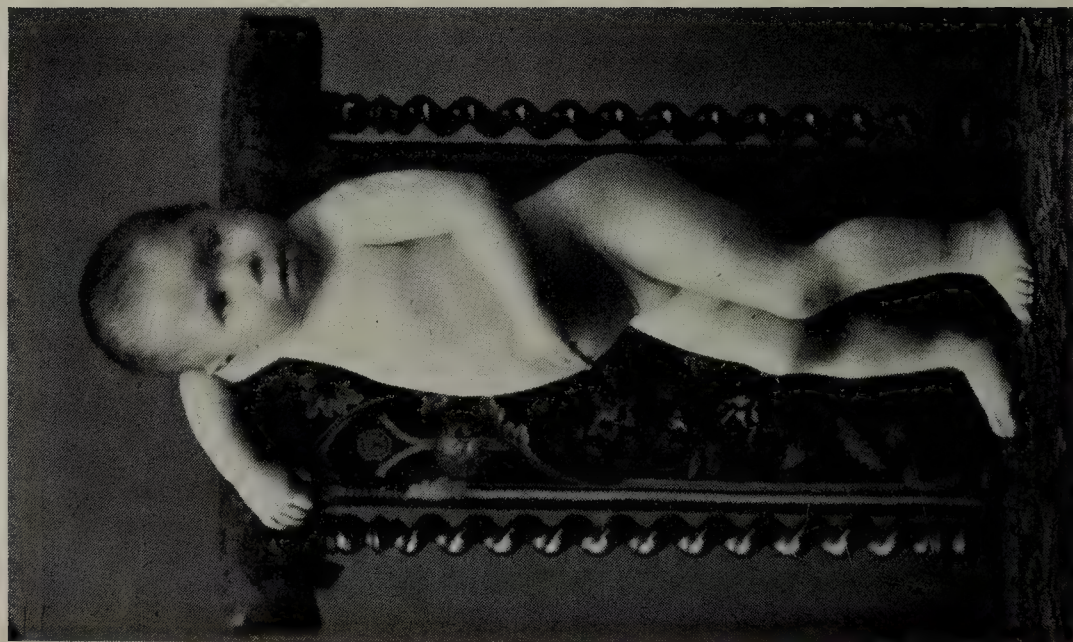


FIG. 162.—Before treatment. Age $4\frac{1}{2}$ years. Height 32 in.



FIG. 163.—After 7 months' treatment. Height $34\frac{1}{4}$ in.



FIG. 164.—After 12 months' treatment. Height $37\frac{3}{4}$ in.

is given regularly to a cretin from infancy onwards, the permanent teeth often appear rather earlier than usual. The appetite is generally much increased by the treatment, and the bowels usually become regular in action.

The growth of the skeleton is perhaps the most striking change of all. It begins at once and proceeds rapidly. The child often gains as much as 2 in. in height within the first two months, and may make as much as 6 or 8 in. or more in



FIG. 165.—Sporadic Cretinism. Same patient as in Fig. 162, 32 years old, with her normal child.

the first year. After that, the rate of growth diminishes and approximates to the normal. In cases where the treatment is begun in early childhood, the return of normal growth affects all the limbs equally. If the patient is an adolescent, however, when the thyroid is first given, the upper limbs grow freely while the lower remain very much stunted.¹ Although the muscular tone of the limbs improves greatly, their finer movements (*e.g.*, those of the hands) are apt to remain clumsy compared to those of a normal child. X-ray examination will

¹ "Variations in and Limits of Improvement of Cretins at Different Ages under Thyroid Treatment," *Brit. Med. Journ.*, 1896, ii., 618.

also reveal great improvement in ossification, but even after many years' treatment this often remains behind the normal rate (Figs. 166 *a* and 166 *b*).

The mental improvement which occurs from thyroid treatment is apt at first to be overestimated by the parents, because the child *looks* so much brighter and his movements are so much livelier than before. Within six months, however, there is unmistakable advance, and this continues and increases, the better nourished brain becoming increasingly capable of good



FIG. 166 *a*.—Skiagram of hand of Cretin aged 11 years, after 6 years' treatment. (Same case as Fig. 152, p. 509.)

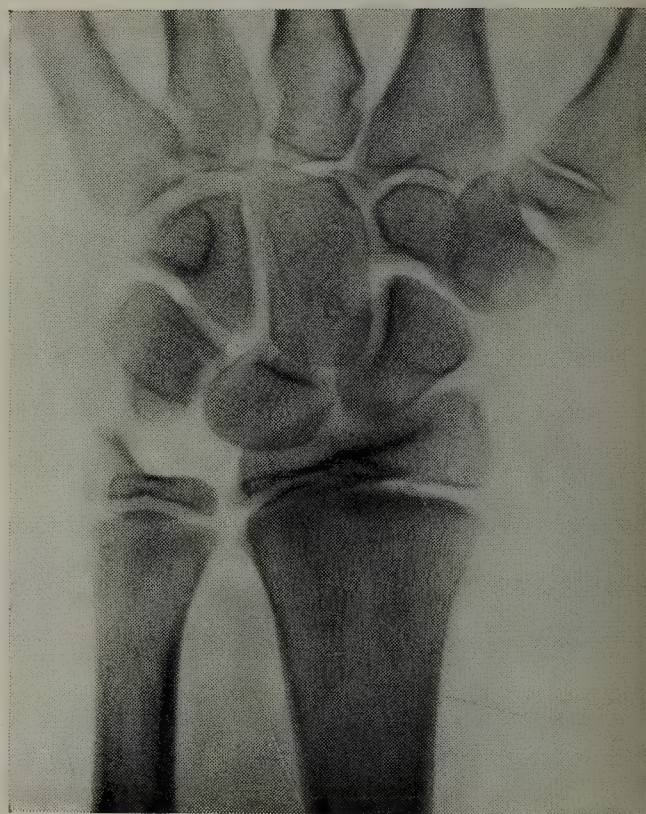


Fig. 166 *b*.—Skiagram of hand of Normal Child aged 11 years.

work. The children grow more inquisitive, more independent and enterprising, and are more inclined to do things. They lose their shy, morose, self-centred disposition, and become happy, playful, childlike, and sociable, but they seldom become quite normal.

In the milder cases the arrears of bodily growth are soon fully made up, the child comes to look normal, and the state of his intellect approaches, and occasionally *quite* reaches, the normal. There is, however, a considerable proportion of cases in which the bodily recovery is more or less complete, but still the child remains an imbecile; and, under these circumstances, the power of speech is often almost or quite in abeyance. In those cases in which the treatment is not begun until later

childhood, the disposition improves and the *capacity for happiness greatly increases*, but the intellectual condition always remains very defective.

Prognosis.—The prospects as to bodily and mental fitness in after-life vary with the degree of the disease present; and also to a considerable extent with the age at which the treatment has been begun, and the regularity with which it is given.

John Thomson records 21 cases in which the treatment was begun early (seven weeks to five years), and which had been kept under observation for many years. Ten of these had become practically normal in intelligence, although most of them were rather slow at work which required manual skill, and some were not very bright mentally. Six out of the 21 continued childish in disposition, and should be classed as feeble-minded or slightly imbecile. The remaining five were more or less severely mentally defective.

Many of the slighter cases are able, on leaving school, to earn a fair wage at various easy occupations. One patient, who has been continuously under treatment for nearly thirty years, is now a very capable wife and housekeeper, and the mother of two quite healthy, well-looking-after children (Figs. 162 to 165).

The Parathyroids.

It has been found that thyroidectomy in man is always followed by tetany, unless one of the parathyroids is left, and that in many animals, though not in all, complete removal of the parathyroids always causes it. When tetany is produced in this way it can only be cured by a successful graft of the parathyroid from an animal of the same species (Schäfer).¹ It is possible, however, to keep the symptoms in abeyance by the production of acidosis from the continuous administration of ammonium or calcium chloride (p. 550).

Escherich² and many other authorities have held that disease of the parathyroid is the cause of the tetanoid condition; but, although there are many pathological and experimental facts which support this theory, there are still difficulties that require to be cleared up before it can be regarded as proved. The subject will be dealt with in the chapter on Spasmophilia (p. 547).

¹ *The Endocrine Organs*, London, 1916, 23.

² *Die Tetanie der Kinder*, Wien u. Leipzig, 1914, 177.

Diseases of the Pituitary Gland.

Hyperpituitarism.—In rare cases, acromegaly begins in later childhood. In one instance,¹ for example, a male acromegalic showed symptoms at puberty; and his daughter, at fourteen, was already a well-marked example of the disease. A patient of Fraentzel's² had typical symptoms at eleven; her father also suffered from acromegaly.

Hypopituitarism.—Deficient pituitary secretion is believed to be the cause of *Fröhlich's Syndrome* or *Dystrophia Adiposogenitalis*.³ This condition is usually seen in older children. Its

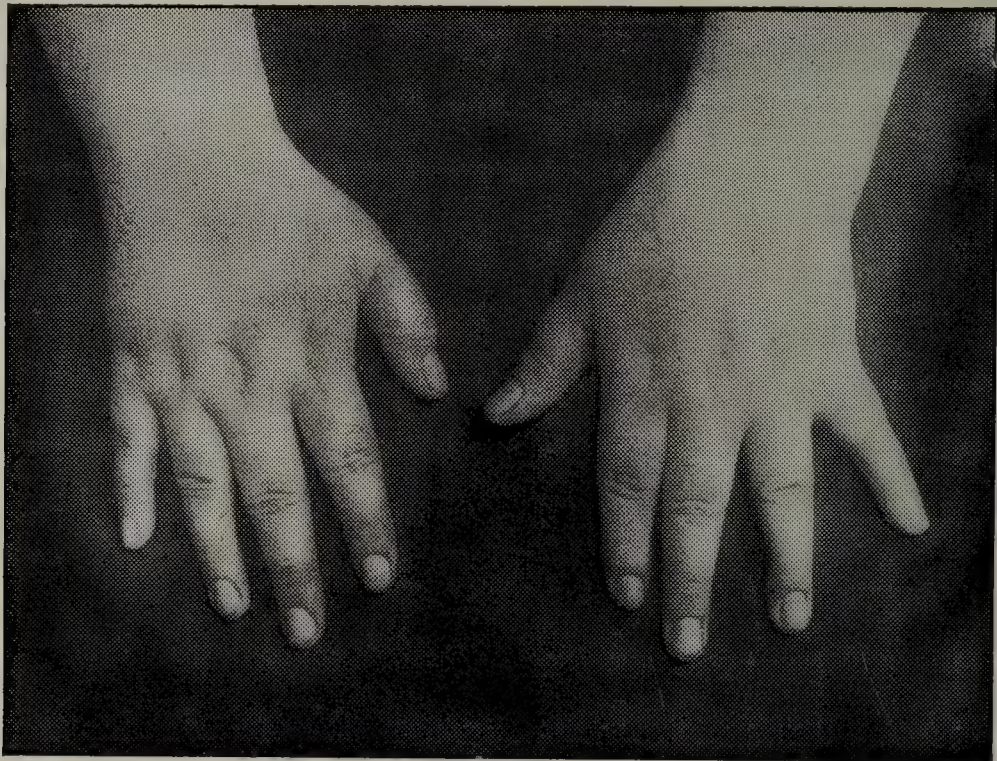


FIG. 167.—Hands in Hypopituitarism.

chief manifestations are obesity, affecting the trunk mainly, peculiar fat hands with conical fingers (Fig. 167), deficient stature, an infantile condition of the genital organs, and some degree of mental defect. There is also usually headache, with bilateral hemianopsia and other signs of a tumour in the region of the hypophysis.

The condition may be due to a tumour of the pituitary, but similar manifestations have sometimes been observed in cases where the gland was found normal on microscopical examination. A striking improvement under treatment with

¹ Franchini and Giglioli, *Nouv. icon. de la Salpêtr.*, 1908, xxi., 325.

² Virchow, *Illust. Med. News*, 1889, ii., 241.

³ Fröhlich, *Wien. klin. Rundschau*, 1901, xv., 883.

pituitary substance has rarely been recorded in these cases. An X-ray examination of the cranium often, though not always, shows enlargement of the sella turcica.

Disease of the Pineal Gland.

Tumours, especially teratomata, are found in the pineal gland in rare cases in childhood. The main symptoms are obesity, remarkable growth of hair in the pubic and other regions, with precocious development of the genital organs, mental precocity or mental defect, and sometimes extreme sleepiness. The symptoms usually develop in early childhood—before nine years old. The condition is distinguished from pituitary disease by the state of the genital organs and by the character of the cerebral symptoms. Its cerebral symptoms also distinguish it from cortical hypernephroma.

Diseases of the Suprarenal Glands.

Addison's Disease is rare in early life, but it occasionally occurs as a symptom of generalised tuberculosis in older children. Absence or defective development of the suprarenals is found in most cases of anencephaly.

The medulla and cortex of the suprarenals are distinct organs; and, when the gland becomes diseased, the symptoms differ entirely according to which of them is involved.

Malignant Medullary Hypernephroma.¹—This is either a neuroblastoma or neurocystoma, most frequently the former, and especially during childhood. It is characteristic of the clinical course of these tumours that the earliest signs of their presence are generally connected with the spread of secondary metastatic deposits in the area of the lymphatic system of the side affected, and do not consist in suprarenal signs or symptoms. At first, the child is vaguely ill and may complain of pains in the lower limbs and have a mitral systolic murmur. A very important early sign is the occurrence of hæmorrhages in the region of the orbit of the affected side, like those seen in infantile scurvy. This is apt to be followed by proptosis from a tumour growing in the wall of the orbit, and tumours may also form on the temporal and other cranial bones, and sometimes on the ribs,

¹ R. Hutchison, *Quart. Journ. Med.*, 1907-8, i., 33; and R. S. Frew, *ibid.*, 1911, iv., 123.

liver, lungs, and thoracic duct (Figs. 168 and 169). The brain itself is not involved, but increased cranial tension, causing headache and optic neuritis, is usual and the case may be diagnosed at first as tumour of the brain. The temperature may or may not be raised; the leucocyte count and the blood pressure are normal.

The patients are mostly under four years old, and the disease, which is always fatal, may last from twelve days to eighteen months (Frew).



FIG. 168.—Metastasis in Temporal Bone in case of malignant disease of medulla of suprarenal gland (Hutchison's type).

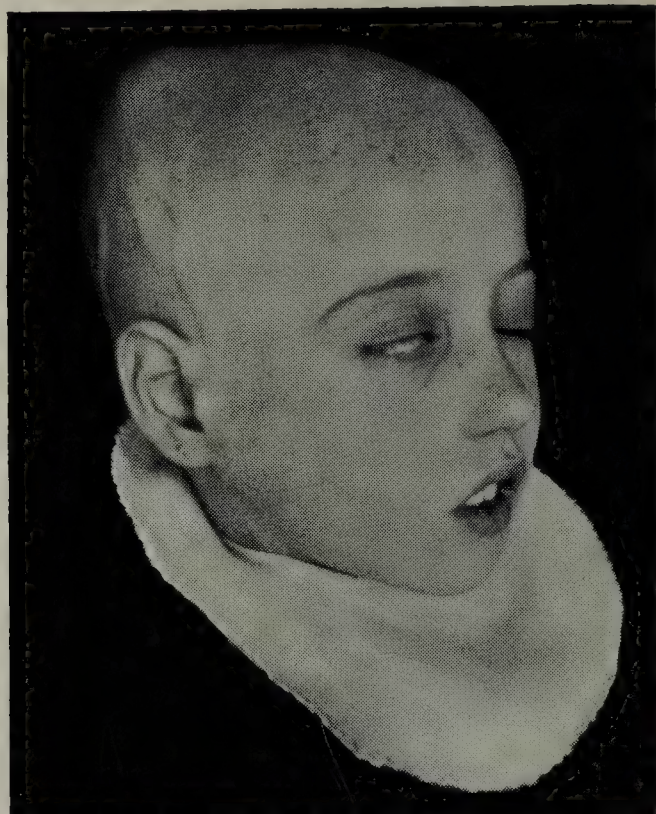


FIG. 169.—Metastases in Parietal Bone and in Ramus of Lower Jaw with right-sided facial paralysis in case of malignant disease of medulla of suprarenal gland (Hutchison's type).

Cortical Hypernephroma.—Tumours in the suprarenal cortex may be simple adenomata or adenomata which have become carcinomatous with metastases. They may produce two varieties of symptoms. In one of these—the *Infant Hercules Type* (Parkes Weber)—which has only been found, as yet, in boys, there is excessive growth of the body, great muscular development, hirsuties, and precocious development in size and function of the sexual organs, with no great obesity.¹ In the other, which usually develops about the second year of life, in either girls or boys, the chief symptom is obesity, sometimes

¹ Dingwall Fordyce, *Quart. Journ. Med.*, 1929, xxii., 557.

with hirsuties, but with no premature development of the sexual functions. The distribution of the fat resembles that seen in adults, being most on the cheeks, neck, and body, and less on the limbs. Although these are the characteristic symptoms of tumour of the suprarenal cortex, in some cases in which they are present nothing abnormal has been found in these glands.

Hæmorrhage into the Suprarenals.—Bleeding into one or both of the suprarenals is frequently found in new-born children as a result of birth injury. It sometimes occurs in children of a few days old from septicæmia; but the most interesting type of the disease is that which occasionally sets in in children during the first year of life as part of a rapidly fatal illness. The disease sometimes occurs as an epidemic, though there is no other reason to believe it to be infectious. The symptoms set in suddenly with severe abdominal pain, high fever, and sometimes vomiting and diarrhœa. Scattered purpuric spots may appear over the surface of the body. Convulsions are common, and death occurs usually within twenty-four hours, with quick pulse and respiration, and general collapse. No treatment has any effect.

The Pancreas.

Affections of the pancreas are considered in this chapter because the gland possesses an internal as well as an external secretion. The former governs carbohydrate metabolism and its absence or deficiency is the cause of diabetes mellitus, whereas the latter is essential for the complete digestion of all the proximate principles of the food—protein, fat and carbohydrate (p. 129). The gland, too, it should be noted, is in close proximity to many important structures. It is thus evident that disease of the pancreas may be manifested in many directions.

Abnormalities in shape and situation of the pancreas may be responsible for several pathological manifestations. The gland may completely surround the duodenum (the so-called *annular pancreas*) and compress the gut so as to lead to stenosis. Accessory pieces of pancreatic tissue when situated in the wall of the small intestine may determine an intussusception.

Occasionally the pancreatic duct suffers obliteration along with the bile-ducts in that inexplicable congenital disease—

obliteration of the bile-ducts (p. 364).¹ When the pancreatic duct is involved, not only is fat absorption interfered with, but also fat-splitting, which results principally from the action of the succus pancreaticus, so that the proportion of neutral fat in the fæces is abnormally high (50 per cent. or more).

As neutral fat cannot be absorbed one can understand how nutrition may be seriously interfered with in disease of the pancreas. Bramwell,² in 1902, described a variety of *infantilism* associated with a pancreatic defect (p. 27). Since then similar cases have been described by other writers, the pancreas being incriminated as the cause from the passage of loose stools containing a high proportion of neutral fat. Langdon Brown³ has recorded an example of this condition in a congenitally syphilitic boy of sixteen years who had the appearance of a boy of eight to ten years. Disease of the pancreas in this case was verified at autopsy.

Disease of the pancreas is, however, not so common in childhood as it is in adult life. This is probably due to the fact that much of the pancreatic disease is dependent on mischief in the gall-bladder and ducts, and this is relatively infrequent in the early years of life. Nevertheless, all varieties of disease of the pancreas may occasionally be met with.

Pancreatitis, both acute and chronic, may occur. The acute variety is rarely suppurative but it may be so as a complication of pyæmia, scarlatina, and enteric fever. Non-suppurative pancreatitis occurs as a complication of most infections, but most notably in association with mumps. The tendency to the increase of glycosuria in diabetes mellitus during the course of any infection is evidence of the frequent implication of the pancreas during the course of infective conditions. The probable reason for its infrequent detection in the normal individual is that Nature invariably supplies an amount of tissue which can function in excess of the minimal needs. A peculiar type of inflammation called *hæmorrhagic pancreatitis*, and believed to be dependent on auto-digestion of the gland, is occasionally met with in the child.

The *symptoms of acute pancreatitis* vary very much in nature

¹ A. F. Hess, *Arch. Int. Med.*, 1912, x., 37.

² B. Bramwell, *Trans. Med. Chir. Soc. Edin.*, 1901-2, xxi., 94, and *Scot. Med. and Surg. Journ.*, 1904, xiv., 321.

³ W. Langdon Brown, *Practitioner*, 1905, lxxv., 233.

and severity. In the milder forms of the condition a transient glycosuria and steatorrhœa may alone be observed. In the severer forms of the disease the clinical picture simulates that of acute peritonitis—abdominal pain, tenderness in the epigastrium, vomiting and diarrhœa with collapse. There is usually fever from the beginning, which fact helps to differentiate the condition from intestinal obstruction and perforative peritonitis.

Chronic inflammation of the pancreas may result from syphilis, as noted above, or be a sequel to acute pancreatitis. Occasionally *cirrhosis of the pancreas* may develop spontaneously, and it has been observed in connection with cirrhosis of the liver and spleen—Banti type (p. 474).

Tumours of the pancreas are rare in childhood. The commonest are of the nature of cysts (retention cysts or cystic adenomata). These may form large tumours and protrude from the abdominal wall anywhere between the xiphisternum and umbilicus. Lymphadenoma, adenoma (cystic or solid), lymphosarcoma and sarcoma (round-celled and spindle-celled) have all been met with in childhood. It is important to remember that if the tumour is situated in the head of the gland it may press on the common bile-duct and simulate hepatic disease (obstructive jaundice). One of us (L. F.) has seen this to occur in the case of a lymphadenomatous mass and of a gumma.¹

Diabetes Mellitus

Diabetes mellitus, which is due to some selective disease of the islets of Langerhans of the pancreas, is not common during childhood. It is variously stated that only about 2 to 5 per cent. of all examples of this disease are met with during the first decade of life. In the course of fourteen years (1916 to 1929 inclusive) there were observed at the R.H.S.C., Glasgow, for example, only 30 cases. During the same period, 13,845 medical cases passed through the wards. The disease is specially rare during infancy. Lawrence and M'Cance,² in a recent survey of the literature, were only able to find records of 25 definite cases, and to this list they added one observation of their own. Of the Glasgow series no case was observed during

¹ L. Findlay, *Syphilis in Childhood*, London, 1919, p. 70.

² R. D. Lawrence and R. A. M'Cance, *Arch. Dis. Child.*, 1931, vi., 342.

infancy, and only one during each of the second and third years of life. The rest of the cases were equally distributed throughout the other years of life up to thirteen years. This points to a relatively increased prevalence during later childhood, since the age incidence of the patients in hospital steadily fell from 30 per cent. during the first year to 5 per cent. during the eleventh and twelfth years. Priesel and Wagner¹ state that the incidence of diabetes during childhood shows a peak about the sixth year and another about the twelfth year.

Boys are more frequently attacked than girls: of the above mentioned 30 cases, 19 were males and 11 females. Jewish children are said to be specially susceptible. This was not evident in the Glasgow series, in spite of the fact that there is a considerable Jewish population in that city. Of Priesel and Wagner's 58 examples, 18 per cent. were of Jewish stock. A history of diabetes in the ascendant is obtained in many cases. In one of the Glasgow series the father was a diabetic. The condition has been observed in each of twins.

The **etiology** of diabetes is not known. Although infections of all kinds apparently damage the islets of Langerhans, as is evidenced by the lowering of the sugar tolerance if some infection supervenes during the course of the disease, seldom in our experience does an infective process initiate the condition. Very occasionally a recent history of some infection, *e.g.*, influenza, mumps, and whooping-cough, is obtained; but it is always possible that the condition had been pre-existent, and was only brought to light through the consequent further impairment of the pancreatic function. Boyd² of Toronto, however, ascribes the onset to an infective process, usually in the tonsils, in 18 out of 32 cases.

Diabetes mellitus is seldom an accidental finding during childhood. **Symptoms** are generally present, particularly polydipsia and polyuria. The polyuria may cause bed-wetting and be the reason for advice being sought. Excessive appetite and wasting are also frequent manifestations, but itching of the skin, ocular conditions, disease of the teeth and nervous phenomena, so common in adult life, are usually absent in the child.

The *diagnosis* of diabetes mellitus as a rule presents no

¹ R. Priesel and R. Wagner, *Ergeb. d. inn. Med. und Kinderheil.*, 1926, xxx., 552.

² G. L. Boyd, *Arch. of Pæd.*, 1924, xli, 291.

difficulty. The symptomatology (gradually increasing polydipsia, polyuria, and emaciation), and the presence of abundant sugar in the urine, seldom leave any room for doubt. It must be remembered, however, that glycosuria *per se* is no criterion of diabetes. In certain individuals sugar readily appears in the urine because of the abnormally low renal threshold (increased permeability of the kidney to sugar). This condition is called *renal diabetes*; but it must not be forgotten that true diabetes has developed in a patient the subject of this peculiarity.

Glycosuria may also appear in a variety of diseases. It is not uncommon in gastro-enteritis during infancy; it may be present in various cerebral conditions (meningitis and tumour), due no doubt to interference with the glycogenic centre in the medulla, and as an accompaniment of ketosis.¹ It may also occur during the course of many infections in consequence of an accompanying pancreatitis (p. 524), and thus only differ from that met with in true diabetes in that it is transient. One must always remember in this connection, however, that even in true diabetes in the young, glycosuria may completely disappear for a time.

Changes in the cerebro-spinal fluid and the presence of optic neuritis would help in eliminating diabetes and indicate a cerebral lesion. A sudden onset of coma, or other grave manifestation, without any premonitory symptoms of the disease, would almost certainly exclude diabetes, and hence ketosis with acidosis, which is always an acute illness, would be easily differentiated from diabetic coma. In the case of some acute infections it may be difficult to say at first whether the glycosuria is due to a transient pancreatitis or the condition is true diabetes brought to light by the infection. Time alone may declare the true state of matters. Mention must also be made that the sugar may not be glucose but lactose: this is not very uncommon during infancy. The diagnosis here rests on the decision, by the aid of the fermentation test, of the exact nature of the reducing substance.

In diabetes the underlying *pathogenesis* is a disturbance of carbohydrate metabolism. There is a diminished ability to utilise sugar so that it accumulates in the blood, and, rising above the renal threshold, flows over into the urine. At the same time the complete combustion of fat is hindered, so that

¹ M. L. Gilchrist, *Arch. Dis. Child.*, 1932, vii., 169.

the intermediate products of the acetone series appear (p. 551), and it is these substances which are in some way associated with coma. It might thus seem that tests of sugar tolerance would be the most reliable method of recognising the disease. This indeed is true, but it must be borne in mind that it is just by disturbing carbohydrate metabolism that some of the above mentioned diseased processes simulate diabetes, and hence special tests conducted while these pathological conditions are active will be of no material help. Consequently the diagnosis, especially during the early stages, may present great difficulty and for a time a definite decision may be impossible. Prolonged observation alone may decide the issue.

Sugar tolerance is estimated in one of two ways: (*a*) by noting how much sugar can be ingested without glycosuria developing, and (*b*) by observing the behaviour of the blood sugar after the ingestion of a definite amount of carbohydrate.

It is generally stated that evidence of increased tolerance is the ability of an individual to ingest 100 or more grammes of sugar without subsequent glycosuria. But since the healthy subject (child as well as adult) can consume much larger amounts, indeed to the point of nausea, without sugar appearing in the urine, it is difficult to understand what is meant by the term increased tolerance. In any case, such a test can have no pathological significance. In the presence of any marked reduction of sugar tolerance, glycosuria may appear after the ingestion of very small amounts. This, however, may be due to a lowered renal threshold, already referred to, and not to a disturbance of carbohydrate metabolism, so that one must not necessarily deduce that there is disease of the pancreas or other endocrine gland.

More exact information regarding the state of carbohydrate metabolism is obtained from observations on the blood sugar content. It is only in this way that those examples of glycosuria due to a lowered renal threshold can be definitely recognised. It may be here mentioned that Dr Gilchrist¹ has shown that the renal threshold is higher in the child than in the adult, and probably lies about .230 grammes per cent. instead of the reputed .180 to .200 grammes per cent. Thus glycosuria with a less value for the blood sugar than .230 grammes per cent. points to renal diabetes. It must, however, be appreciated that

¹ M. L. Gilchrist, *Arch. Dis. Child.*, 1929, iv., 129.

there prevails the greatest variations within the limits of normality, and hence care must be exercised in drawing conclusions from observations on the blood sugar. It must also not be forgotten that the blood sugar will show deviations from the normal, no matter what the cause of the disturbance of carbohydrate metabolism, so that the decision by this means of the exact cause of the glycosuria may not be rendered any easier.

The blood sugar content normally fluctuates between fairly definite limits. After a meal containing carbohydrate the blood sugar rises and reaches a maximum of $\cdot 180$ grammes per cent. within one-half to one hour and thereafter declines, reaching by the end of four to eight hours its lowest level, the so-called fasting level. This is generally taken to lie between $\cdot 070$ and $\cdot 120$ grammes per cent., but Dr Gilchrist,¹ working at the R.H.S.C., Glasgow, found the fasting level, even after such long periods of starvation as eight to fifteen hours, to vary in the normal child between $\cdot 067$ and $\cdot 166$ grammes per cent. Other observers² have shown the same to be true of the adult. In untreated diabetes the fasting level is usually much in excess of this. In severe cases it may register $\cdot 270$ to $\cdot 300$ grammes per cent. and in diabetic coma $\cdot 500$ grammes per cent. However, in mild examples of the disease the fasting level may be within normal limits, and in order to recognise these less severe cases it is necessary to observe the course of the blood sugar after its oral administration.

In the normal adult, after a standard dose of 50 grammes of glucose, a more or less constant blood sugar curve is obtained. The blood sugar rises almost immediately after ingestion and reaches its maximum of $\cdot 180$ to $\cdot 200$ grammes per cent. within one-half to one hour and thereafter falls, reaching its original fasting level within one and a half to two hours. Many workers recommend the same dose for the child, excepting perhaps in the case of the infant, but there is no doubt that with such an amount apparently pathological and hence misleading results are often obtained. It would only seem rational to make the dose commensurate with the age of the individual, and when this is done, using amounts equivalent to 1 gramme per kilo of expected body weight, a more uniform curve is obtained. This curve corresponds closely to that described for the adult,

¹ *Loc. cit.*

² H. Gray, *Arch. Int. Med.*, 1923, xxxi., 241.

excepting that the initial rise is higher and the fall to the fasting level somewhat slower. With this proportionate dose Dr Gilchrist¹ found that in the normal child the maximum rise varies between .141 and .204 (average .173) grammes per cent. and occurs within one-half to one hour after ingestion. In only 50 per cent. of the children, however, even with this moderate dose, was the fasting level reached within two hours, but in most of them this had occurred within two and a half hours.

In diabetes, on the other hand, a very different curve is usually obtained. The blood sugar rises higher (always above .200 grammes per cent.) and it may not return to the fasting level till three or four or more hours later. As already pointed out, the same behaviour of the blood sugar is observed in examples of disturbed carbohydrate metabolism due to pancreatitis, cerebral disease, and ketosis, but in renal diabetes a normal curve is obtained, and glycosuria appears while the blood sugar is at a level below .180 to .230 grammes per cent.

Hamman and Hirschmann,² and at a later date Staub³ and Traugott,⁴ have recommended a modification of the above test, the result of which they consider more easy of evaluation. This modification consists in the administration of glucose in small doses (10 to 20 grammes) repeated every half to two hours, *i.e.*, before the blood sugar has had time to return to the fasting level. Traugott states that in health and in renal diabetes the blood sugar may not show a fresh rise after the second dose, or if such does occur it is very slight, and that after the third dose of glucose the blood sugar invariably returns to its fasting level. In diabetes mellitus, on the other hand, the rise after the second dose is considerable, and so long as the doses of glucose are repeated, he holds that the abnormal level of the blood sugar is maintained. The published records, however, do not to our mind show this sharp distinction, and, in any case, this technique cannot eliminate disturbances of carbohydrate metabolism accompanying infections, cerebral lesions, and ketosis.

The **prognosis** of diabetes in childhood has been quite

¹ *Loc. cit.*

² L. Hamman and I. I. Hirschmann, *Bull. Johns Hopk. Hosp.*, 1919, xxx., 306.

³ H. Staub, *Zeit. f. klin. Med.*, 1921, xci., 58.

⁴ K. Traugott, *Klin. Wochenschr.*, 1922, i., 892 and 2384.

changed since the introduction of insulin therapy. Prior to the insulin days the disease was as a rule rapidly fatal (the duration of life was seldom longer than two years after its inception) and children developing coma usually died. Since the introduction of insulin this state of matters has been completely changed. The child in coma can generally be saved, and his ability to utilise carbohydrate brought up to a normal level, so that, with continuous treatment, he may lead a more or less normally active life. One boy, whom one of us (L. F.) saw in 1920 at the age of eight years, has, with continuous treatment, attended school regularly and passed into the University, and is now at the age of twenty years a medical student. There is never, however, any question of cure. Priesel and Wagner¹ state that the course of the disease is slowly progressive and that cases which at first can be controlled by dietetic measures ultimately require insulin. In our experience, although a cure has never been observed, the condition does not necessarily get worse. Rarely, for a time at least, the child's tolerance for carbohydrate may improve, and that quite apart from the recovery after an intercurrent infection. To Drs Fleming and Morris we are indebted for the following analysis relative to this point of the cases which have passed through the R.H.S.C., Glasgow.

Behaviour of Sugar Tolerance in Diabetes in the Child.

	No.	Average Age at Onset.	Average Age now.	Average Age at Death.	Average Duration.
Improved tolerance .	3	3.5 yrs.	7.6 yrs.	...	4.0 yrs.
Stationary tolerance .	5	6.5 „	11.2 „	...	4.5 „
Diminished tolerance	9*	8.5 „	13.0 „	...	4.4 „
Dead	13	8.4 „	...	11.5 yrs.	3.1 „

* In these children the dose of insulin per gramme of carbohydrate is increasing.

It will be noted in the above Table that it was the very youngest of the patients who showed improvement in their sugar tolerance. This is interesting in view of the recent observation of Lawrence and M'Cance² of definite recovery in two examples affecting children under one year. On the introduction of

¹ *Loc. cit.*

² *Loc. cit.*

insulin therapy it was hoped that in this way rest would be provided the islets of Langerhans, and thereby perhaps permit of their regeneration. There is, however, at present no unequivocal evidence that this does occur.

Treatment.—Treatment consists in supplying the internal secretion of the pancreas, which is insufficient for the amount of carbohydrate necessary for ordinary life.

Whenever the condition is recognised it is advisable, even in the mildest case, that the child be put to bed, either in a nursing home or a hospital, where blood sugar estimations may be carried out and the patient's ability to utilise carbohydrate is measured.

Treatment may be initiated in one of two ways. (a) The child may be put on a very low diet and the urine rendered sugar-free if possible, after which the diet is gradually increased till sugar appears in the urine. If this diet is not sufficient to supply the child's requirements, then additional carbohydrate with insulin sufficient to cover the excess carbohydrate (one unit of insulin for every two grammes) must be supplied. In estimating the amount of carbohydrate in the diet it should be borne in mind that half of the protein is burned up as carbohydrate and must be considered as such. (b) The child may be given his optimum of food from the beginning. The daily output of sugar in the urine is estimated, and an amount of insulin injected which corresponds to one unit for every two grammes excreted.

Morris¹ considers the second method preferable if "there is marked ketonuria, because it is very unwise to restrict the food of a child when this is present, since children are very susceptible to acidotic conditions and coma may easily be precipitated. Otherwise the first or 'crescendo' method of treatment is to be preferred, and for the following reasons:— (1) It enables one to determine the carbohydrate tolerance before giving insulin. (2) It permits the gradual storage of glycogen in the liver, thereby lessening the chance of hypoglycæmia. This occurrence should be avoided if possible, particularly in the early stages of insulin treatment, lest it irreparably undermine the patient's and the parents' confidence in insulin. With small initial doses the parents are gradually introduced to insulin treatment. (3) By beginning with large doses of insulin there may be a tendency to over-feeding."

If it is desired to practise the former method, the following

¹ Noah Morris, *Glasg. Med. Journ.*, 1932, N.S., xxxvii., 321.

rules should be observed. Roughly the diet should supply the basal requirements, *i.e.*, 20 to 25 calories per kilo body weight. Thus for a child of six years weighing 20 kilos an amount of food representing 400 to 500 calories per day is necessary. The protein, as also the fat, should at first be given in amounts equal to one gramme per kilo, which represents a total of 260 calories. The remaining 140 to 240 calories are supplied by carbohydrate, which means an amount varying between 35 and 60 grammes. When once the urine is sugar-free the diet can be gradually increased, remembering, however, that it is wise to err on the side of under-feeding the diabetic. Yet under-feeding must not be overdone, as it will only tempt the child to obtain, by hook or by crook, an amount of food which satisfies. Generally it may be said that 25 per cent. of the caloric requirements should be supplied by protein, 50 per cent. by fat, and 25 per cent. by carbohydrate. If the sugar cannot be rendered sugar-free with the above minimal diet, then insulin must be supplied in amounts to cover that lost in the urine (one unit of insulin for every two grammes). If the case is mild a larger proportion of carbohydrate may be given.

The requisite diet can be easily prescribed with the help of the two following tables. In one table (Table A) there is shown for different ages the recommended caloric intake (which is the basal requirements plus 50 per cent.), the best proportion and amounts of the various proximate principles as well as the maximum proportion and daily amount of protein, which latter must not be exceeded. In the other table (Table B), compiled after Lawrence, there is given the relative amounts of protein, fat, and carbohydrate in various common foodstuffs. The foods containing carbohydrate are underlined by a red line and those containing protein and fat by a black line—hence this system is generally called the “line diet,” and is one that can be easily carried out by the patient or his mother. It is advisable always to balance a red line by a black line, the two combined representing 120 calories. It is also advisable to supply the bulk of the food in two meals, the requisite amount of insulin being given half an hour beforehand. By this procedure the discomfort of the injection is diminished as much as possible. Seldom if ever is one injection per day successful, and in some cases it may be necessary to distribute the insulin throughout the day (*e.g.* three injections) in order to keep the urine sugar-free.

A.—Maximal Amounts of Proximate Principles advisable in Diet of Diabetic Child.

Age in Years.	Expected Weight in kilo.	Maximum Protein in grammes per kilo.	Calories required per Day. (Basal rate + 50 per cent.)	Approximate No. of Black and Red lines of Line Diet supplying the foregoing.*	Distribution of Proximate Principles in foregoing Diet.			Distribution of Calories in foregoing Diet.		
					Protein.	Fat.	Carbo-hydrate.†	Protein.	Fat.	Carbo-hydrate.
4—	16.0	3.8	950	8.0	60.0	40.0	240	540	160	940
5—	18.5	3.5	1000	8.5	64.0	42.5	256	576	170	1002
6—	20.0	3.4	1060	9.0	67.5	45.0	270	608	180	1058
7—	22.2	3.3	1110	9.5	71.5	47.5	286	644	190	1120
8—	24.0	3.1	1170	10.0	75.0	50.0	300	675	200	1175
9—	27.0	2.9	1220	10.5	79.0	52.5	316	711	210	1237
10—	29.5	2.8	1270	11.0	82.5	55.0	330	743	220	1293
11—	32.0	2.7	1320	11.5	86.5	57.5	346	779	230	1355
12	35.0	2.6	1380	12.0	90.0	60.0	360	810	240	1410

* One red line must always be balanced by one black line in diet: more than one black line to each red line may be given in less severe cases.

† This amount of carbohydrate can of course be exceeded depending on severity of condition.

B.—Line Diet after Lawrence, showing Relative Amount of Proximate Principles in Various Foodstuffs.

Carbohydrate, 5 grammes.		Protein, 7½ grammes.		Fat,† 7½ grammes.
(Black Lines*)	Oz.	(Red Lines*)	Oz.	Oz.
Cabbage or Greens	6	One Egg	
Cauliflower or French Beans	6	Lean Bacon	I	
Spinach, Asparagus	6	Ham	I	
Lettuce (raw)	6	Kipper	I½	¼
Brussel Sprouts	5	Herring	I	¼
Turnip	5	Lean Meat or Mutton	I	¼
Cucumber, Celery or Cress	5	Lean Lamb or Veal	I	¼
Tomato (raw or cooked)	4	Chicken or Duck	I	
Carrot, Leeks or Artichoke	4	Tongue (tinned)	I	
Milk	3½	Liver	I	¼
Swedes or Radishes	3½	Kidney or Tripe	I½	¼
Onions or Green Marrow	3½	Rabbit	½	¼
Parsnips	2	Cheese	¾	
Orange or Strawberries	2	White Fish	I¼	¼
Apple, Pear or Raspberries (raw)	1½	Sardines	I	¼
Bread			
Oatmeal (raw) Biscuits or Toast	¼			

Each “black” line of diet chart equals 5 grammes of carbohydrate and each “red” line equals 7½ grammes of protein and 7½ grammes of fat. One “black” and one “red” line together equal 120 calories.

* When this chart is supplied to patient each foodstuff is underlined by either the appropriate black or red line.
† Fats are :—Meat fats, Dripping, Butter, Margarine and Olive Oil. Thick Cream, if used, to be given in twice above amounts.

The urine should be examined at least twice a day and a chart kept. This the older patients or the parents can be taught to do quite satisfactorily.

Mention has already been made of the further lowering of the sugar tolerance during the course of any intercurrent infection. This is well exemplified in the following two cases

Deterioration of Sugar Tolerance during Infection.

Case.	Weight.	Units of Insulin required.	Blood Fasting Sugar. (grammes per cent.)	Urine.		Condition.
				Sugar.	Acetone.	
A	17.2 kilo	16	.070	nil	nil	Well.
„	17.0 „	22	.225	++	+++	Septic wound of hand.
B	21.5 „	20	.081	nil	nil	Well.
„	21.2 „	44	.124	++	— +	Influenza.

consequent on a septic wound of the hand in the one and an attack of influenza in the other. Thus at the time of any intercurrent illness special care must be observed.

Diabetic children seldom die of any intercurrent disease. Death usually results from coma. Of the thirty children comprising the Glasgow series thirteen died, and in twelve instances from coma. Twelve of the children died at home and were therefore not under proper supervision. In the presence of coma an attempt must be made to bring about a cessation of the malcombustion of fat and the production of ketones. This is done by the administration of carbohydrate amply guarded by insulin. Large doses of insulin (10 to 30 units) should be administered at once and repeated every two or three hours, the amount and frequency being indicated by the general condition of the patient and the rate of disappearance of the acetonuria. The acidosis produced by the ketones may be counteracted by alkali, *e.g.*, 10 to 15 grains of bicarbonate of soda every four hours. This, however, is not essential, as the disappearance of the ketones itself removes the cause of any tendency to acidosis. In diabetic coma dehydration is always a marked feature, and hence it is a wise procedure to administer saline by rectum or subcutaneously.

Within recent times the advisability of continuous observation of chronic illness has become apparent, and it is now the general practice in quite a few diseases. Clinics for this purpose were first instituted in connection with orthopædics, at a later date for poliomyelitis, still later for rheumatism, and lastly for diabetes. One such clinic for the child diabetic was instituted at the R.H.S.C., Glasgow, in 1928, and has been followed by great benefit, as is shown in the analysis of the material by Dr Fleming.¹

Effect of Continuous Supervision of the Diabetic Child.

	No. of Cases.	Improved or I.S.Q.	Diminished Tolerance.	Dead.
Pre-clinic (1922 to 1928) .	10	1	...	9
Clinic (1928 to 1932) .	20	7	9	4

¹ G. B. Fleming, *Glasg. Med. Journ.*, 1932, N.S., xxxvii., 314.

The clinic has a value in many directions.¹ Through its medium the parents and the children are gradually trained in the conduct of their particular case (examination of urine and weighing of food); they gain confidence in the treatment as well as appreciate the necessity for it, and thus there results better co-operation between doctor and patient, which is a *sine qua non* for success in this most distressing malady.

¹ Much useful information regarding the conduct of such a clinic will be found in an interesting survey by Dr Noah Morris, which appeared in the *Glasgow Med. Journ.*, 1932, N.S., xxxvii., 321.

CHAPTER XXII

SPASMOPHILIA OR TETANY¹

Spasmophilia is a disturbance of nutrition of as yet unknown cause, characterised by hyperexcitability of the nervous system and specially prevalent during the first three years of life. It is a frequent accompaniment of rickets, is occasionally associated with severe gastro-enteritis, or any marked alkalotic state of the blood such as occurs in hypertrophic pyloric stenosis or post-encephalitic hyperpnœa.

The disease may be present in either a *latent* or a *manifest* form. In the latent form it is demonstrated by the presence of *electrical* and *mechanical hyperexcitability* of the peripheral nerves and a *lowered blood calcium*. In fact, it is these various phenomena which are our best clinical tests of its presence.

(a) **Erb's Phenomenon**, which consists in an abnormal excitability of the peripheral nerves to galvanic stimulation, is generally the earliest indication of spasmophilia to develop, and it is also that sign which is most constantly present. Normally the cathodal opening contraction requires a current of 8 to 10 milliamperes, but in tetany this contraction can be elicited with a current of less than 5 milliamperes. The fact that the anodal opening contraction is elicited with a less current than the anodal closing contraction is not, as is often supposed, characteristic of tetany, since the same qualitative change is frequently observed in health.²

To apply the test, the peroneal nerve-muscle group may be used, the positive electrode being placed over the upper abdomen and the negative over the peroneal nerve as it winds over the head of the fibula; or the median nerve or ulnar nerve may be preferred. The skin resistance varies directly in individual instances according to the amount of subcutaneous fat present, and diminishes rapidly as the operation proceeds.

¹ A. F. Hess, *Rickets, Osteomalacia, and Tetany*, Philadelphia, 1929.

² D. N. Paton, L. Findlay, and A. Watson, *Quart. Journ. Exper. Phys.*, 1916, x., 281.

(b) **Chvostek's Sign** or the **Facial Phenomenon** is due to increased mechanical excitability of the nerves, and is the contraction of the facial muscles which ensues on tapping sharply over the facial nerve (*pes anserina*). The contraction resembles that caused by the passing of a galvanic current (Figs. 170 and 171). It is apparent in the frontalis muscle at the root of the nose, in the muscles of the eyelids, and in those of the alæ nasi and mouth. It may be evident in all these situations but in varying intensity; it is, however, often only present at the root of the nose. Sometimes it is more marked on one side of the face than the other, and, indeed, may only be

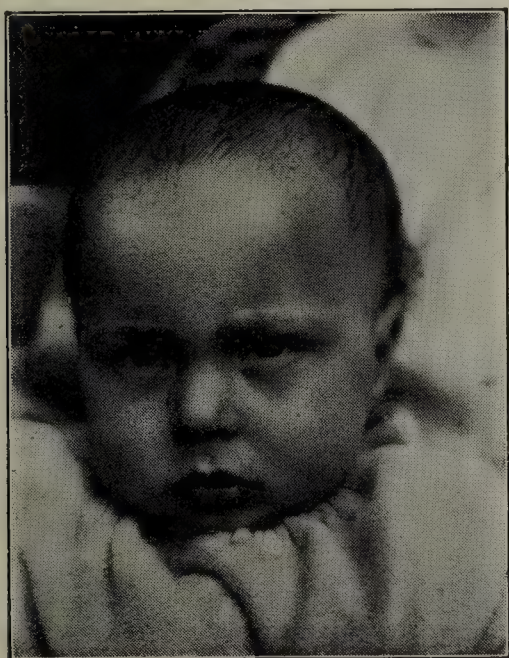


FIG. 170.—Facial Irritability.
Before tapping.

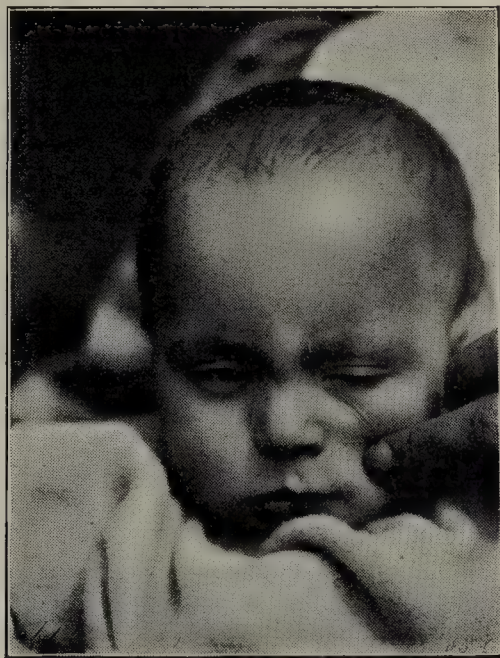


FIG. 171.—Facial Irritability.
On tapping.

elicited on one side. It is important to bear in mind that after a convulsion the hyperexcitability of the nerves disappears temporarily, and hence neither Chvostek's nor Erb's phenomenon can be elicited.

Chvostek's sign must not be confused with the normal infantile lip reflex (p. 683). The former is observed equally during sleeping and waking, in children of any age, and is not a true reflex, but due to direct mechanical stimulation of the nerve. The movements elicited are confined to the side tapped; they are momentary, and are not at all co-ordinated or purposive. When found in young children it probably always indicates the presence of spasmophilia. It is rarely met with in infants under five months old.

The lip-reflex occurs almost exclusively during sleep, is

most active in very young babies, and has the characters of a true reflex. Its movements involve both sides of the mouth, and they are deliberate, co-ordinated, and purposive. In its ordinary form the lip-reflex is a normal phenomenon.

A slight degree of facial irritability is not uncommon in older children (McNeil).¹ S. G. Graham and Grace H. Anderson found it present in 29 per cent. out of 1723 children between five and eighteen years.² It occurs in them sometimes with slight dyspeptic disorders, and frequently apart from any ascertainable disease. In young children it is the most useful diagnostic sign of spasmophilia. In the older ones it has no such significance. A severe degree of Chvostek's sign occurs in older children in some cases of tetany and in progressive dementia.

(c) **Trousseau's Sign.**—This is the name given to the appearance or recurrence of the characteristic tetany posture of the child's hands or feet, during a free interval, when the main artery or nerve of the limb is compressed. In many cases this symptom cannot be obtained in young children; but in some it is very well marked. It is most easily elicited by tying a piece of rubber tubing round the upper arm. It is variously ascribed to anæmia from occlusion of vessels and to direct irritation of the nerves.

(d) **The Blood in Spasmophilia.**—The lowered calcium content of the blood is one of the most delicate tests for the detection of spasmophilia. Normally, the blood calcium (CaO) amounts to between 9 and 11 mgrm. per cent. of the serum, whereas in tetany this is constantly lowered, but not necessarily proportionately to the severity of the disease. In latent tetany it may fall to 7 or 8 mgrm. per cent., and in the active stage to 6, 5, or even 4 mgrm. per cent. This fall in serum calcium has a considerable practical importance since, with the exception of uræmia, it is not met with in any other disease, and hence differentiates spasmophilia from other types of convulsions. Along with the fall in calcium there is usually, though not invariably, a rise in the amount of the inorganic phosphorus (P_2O_5) from 4 to 5 mgrm. per cent., which is the normal level, to 7 or 9 mgrm. per cent.

In the active form of the disease the manifestations are in

¹ C. McNeil, *Edin. Med. Journ.*, 1924, N.S., xxxi., 651.

² S. G. Graham and G. H. Anderson, *Lancet*, 1924, i., 1307.

order of frequency, (1) *laryngismus stridulus*, (2) *convulsions* or *infantile eclampsia*, and (3) *carpo-pedal spasm* (*tetany*). In many children all three manifestations may occur, but in not a few only one or other, or various combinations, of these different evidences may be present. In 100 consecutive cases 69 showed facial irritability, all presented laryngismus, 12 had tetany, and no fewer than 60 were said by their mothers to have had general convulsions.

(1) **Laryngismus Stridulus** (*Spasmus Glottidis*, *Child Crowing*).—This manifestation of spasmophilia is only observed during infancy, and is a spasmodic nervous affection of the respiration which, in its typical form, causes a sudden arrest of breathing for a few seconds, or longer, with the chest in the position of expiration, and is followed by a crowing inspiration. While this is what generally happens, the arrest of breathing may occur with the chest in the position of inspiration, and then no crowing follows. The most striking phenomenon in an ordinary attack is the spasmodic closure of the glottis, but the other muscles of respiration share in the spasm. Laryngismus, therefore, is not to be regarded as merely a local spasm of the glottis.

The symptom, in its typical form, probably only occurs in children who are the subjects of spasmophilia, and the patient has usually been out of sorts for some days or weeks before it sets in; but the attacks often set in very suddenly. The patient, who has been sleeping quietly or playing about as usual, all at once stops breathing, looks scared, and throws back his head with his mouth open. The body and limbs are rigid and the fists clenched. The face is at first cyanotic, and afterwards ashy pale. There may be a short loss of consciousness, and occasionally the attack ends in a general convulsion. After a few seconds the spasm relaxes and there is a loud crowing inspiration. In many of the slight cases a few laboured inspirations, accompanied by crowing, are all that can be observed. In the severe type, the arrest of breathing may be so complete and prolonged that the child dies in the attack. The number of the paroxysms, as well as their severity, varies greatly in different cases. In some there are only a few seizures in the twenty-four hours; in others there may be twenty, thirty, or more. They generally occur more frequently during the night.

Laryngismus also is more frequent in boys, and it often

occurs in several members of a family. As Gee and others have pointed out, it is most commonly met with in this country in the first half of the year. In the hundred consecutive cases above referred to, 81 occurred between January and June, and only 19 between July and December. This is the same seasonal incidence as is observed in the case of rickets (p. 246).

The *immediate cause* of the seizure is generally some emotional disturbance or slight nervous shock. A paroxysm often occurs on awaking from sleep, when the child is suddenly exposed to a draught of cold air, or when he is frightened or annoyed in any way—also during swallowing and straining. The tendency of emotional shocks to bring on a severe paroxysm is a most important matter in practice. One result of it is that children with laryngismus are among the very few cases that are generally better treated at home, even in poor surroundings, than in a hospital ward. In them the comfort and assurance afforded by the mother's arms count for more than the most skilful nursing.

The **diagnosis** of laryngismus usually presents no difficulty. The condition which most closely resembles it is the **nervous holding of the breath**, which is sometimes met with in young children, especially in those who are neurotic and spoiled. Probably, as H. Neumann¹ and Ibrahim² have suggested, these attacks are manifestations of infantile hysteria. The seizure is often brought on by a fit of temper or a sudden fright; and the child, who is generally between two and five years old, finds on trying to scream that he cannot do so. He turns pale, his body stiffens, and he may become cyanosed and lose consciousness. After a few seconds, or at most some minutes, the respiratory spasm relaxes; and, without any crowing inspiration, he begins again to breathe and cry normally. These attacks differ from laryngismus in their lesser severity, in the patients being usually older children, and in the laryngismus not being accompanied by other symptoms of spasmophilia. The condition is generally a trivial one, though in a patient who was the subject of lymphetism the attack might prove fatal. The treatment is that of the hysterical state, and the mother should

¹ "Ueber das Wegbleiben kleiner Kinder," *Arch. f. Kinderheilk.*, 1905, xlii., 99.

² "Ueber respiratorische Affectkrämpfe im frühen Kindesalter," *Zeitschr. f. d. ges. Neurologie u. Psychiatrie*, 1911, v., 3, 388.

be told not to be anxious. As Leonard Guthrie says, "the temperate use of a slipper" is often an excellent remedy.

False Croup should never be mistaken for laryngismus. The patients in the latter are younger; the spasms are shorter in duration; cough, fever, and coryza are absent. In any doubtful case, a history of convulsions and the presence of facial irritability or tetany are strongly in favour of the diagnosis of laryngismus. A low blood calcium would settle the matter (p. 540).

It is important to remember that attacks, which closely resemble those of laryngismus, occasionally occur in atypical whooping-cough in young babies.

If the attacks of laryngismus are at all severe, a guarded **prognosis** must be given until the treatment has begun to take effect. The great majority of cases recover rapidly and completely, but there is always a chance that the next attack may be severe and prolonged, and a possibility that the child may die in it.

(2) **General Convulsions or Infantile Eclampsia.**—Epileptiform convulsions during infancy are most frequently due to spasmophilia. They vary much in severity and type, from a momentary loss of consciousness and rolling of the eyes to prolonged generalised convulsions. The diagnosis of their true nature turns on discovering some other evidence of spasmophilia—a history of laryngismus, a lowered calcium content of the blood, and increased electrical and mechanical excitability of the peripheral nerves, though, as previously mentioned, both these evidences may be absent for some time after a convulsive seizure.

(3) **Tetany or Carpo-pedal Spasm.**—Tetany or the tetanoid state of the muscles is most frequently limited to those of the hands and feet, when it is called carpo-pedal spasm. The hands, owing to spasm of the interossei, assume the so-called "accoucheur's position" (Figs. 172, 173, 174, and 175). The fingers are somewhat flexed at the metacarpo-phalangeal, and fully extended at the interphalangeal joints. They often overlap one another, and the thumb crosses the palm so that its tip touches the middle phalanx of the ring finger (Fig. 175). In young babies the position of the hand is often different, the fingers being merely flexed to an extreme degree, with the thumb either doubled under them or projecting between two

INFANTILE TETANY.

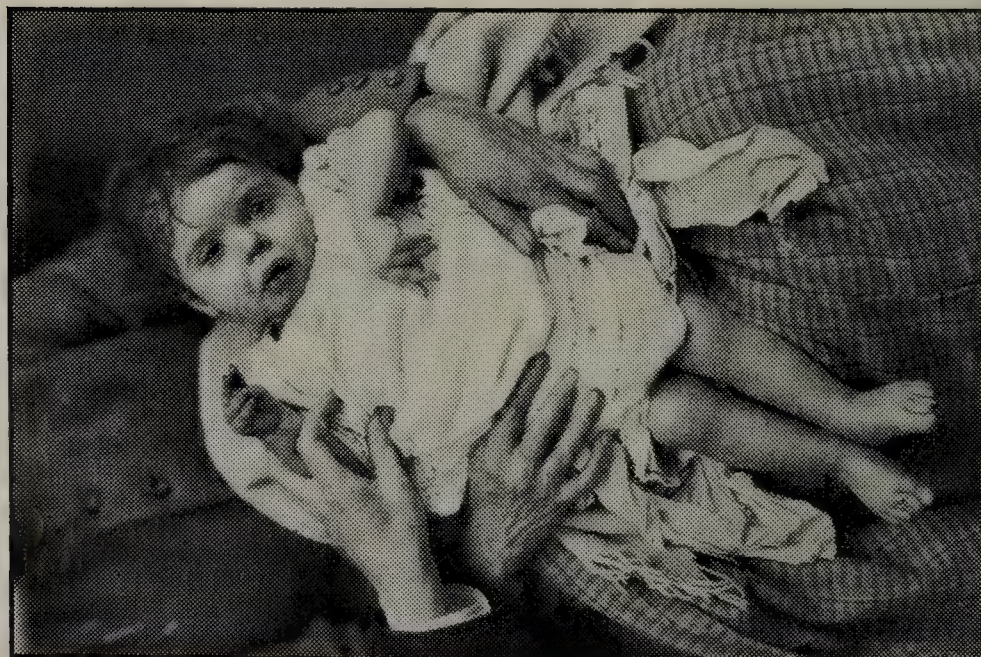


FIG. 172.—Girl of 13 months.



FIG. 173.—Girl of 9 months.



FIG. 174.—Boy of 7 months.

of the proximal phalanges. Various transitional postures are met with, such as that seen in Fig. 173. The spasticity may in severe cases involve the muscles of the wrist and elbow.

The lower limbs are usually affected along with, and sometimes without, the upper. The front part of the sole is contracted so as to produce an antero-posterior furrow, and there is extreme plantar flexion of the toes. In some cases the muscles of the back and neck are involved, causing rigidity of the neck and slight head-retraction. On rare occasions the muscles of the abdomen and even the bladder and rectum



FIG. 175.—Carpal Spasm in boy of 10 years suffering from Tetany.

may share in the spasm; and some writers have described cardiac tetany causing sudden death.¹

Pathogenesis of Spasmophilia.—In the great majority of instances spasmophilia is associated with rickets and by some writers (Kassowitz² and Cheadle³) has been considered merely the nervous manifestation of that disease. There is no doubt that this association is a very real one and must be taken into consideration in any conception of the pathogenesis of the condition. It is this association which is responsible for these diseases possessing the same seasonal and social incidence. There is, however, no strict parallelism between them. Many

¹ J. Ibrahim, *Jahr. f. Kinderh.*, 1910, lxxii., 346.

² M. Kassowitz, *Gesammelte Abhandlungen*, Berlin, 1914, 192.

³ W. B. Cheadle, *Lancet*, 1887, i., 919.

of the most severe examples of rickets never suffer from tetany, which, indeed, would almost seem to show a predilection for the very mildest of cases.

At the present time there are three views held regarding the cause of the condition. These are that it is (*a*) due to calcium depletion; (*b*) to a state of alkalosis; and (*c*) to guanidin poisoning through dysfunction of the parathyroid glands.

(*a*) *Calcium Deficiency*.—The idea of the calcium deficiency theory originated with Sabbatani.¹ He noted that if the exposed cerebral cortex of an animal was bathed with a solution rich in calcium its electrical excitability was depressed, whereas if the calcium were diminished by precipitation with a solution of oxalic acid the electrical excitability was greatly increased. These experiments, along with the knowledge that in rickets there was a loss of calcium, prompted Quest² in 1905 to compare the calcium contents of the brains of spasmophilic and non-spasmophilic children, and finding less calcium in the brains of the spasmophilic children (although this has not been confirmed by subsequent workers), he concluded that the diminution of this sedative salt of the body was the cause of tetany. This view received strong support by the observation of Howland and Marriott³ (1918) that in tetany the blood calcium was diminished, because it appeared that in this way the tissues would in all probability be supplied inadequately. The fact previously mentioned that the fall in the blood calcium is not proportionate to the severity of the spasmophilic manifestations, and the observation⁴ that bleeding and perfusion with saline relieved the condition were, however, two serious objections to the adoption of this theory of etiology. The latter occurrence made it specially difficult to accept such an explanation, as the procedure of bleeding would naturally still further diminish the available calcium. Nevertheless, the reported good effect of the salts of calcium, especially calcium chloride, was considered confirmatory, if not irrefutable, proof of the calcium depletion theory, until it was ultimately (1923) appreciated that the virtue of calcium chloride did not depend on supplying calcium but on its power to induce a state of acidosis, and that for the

¹ L. Sabbatani, *Revist. Sperim. di frenitria*, 1901.

² R. Quest, *Jahr. f. Kinderh.*, 1905, lxi., 114.

³ J. Howland and W. M'K. Marriott, *Quart. Journ. Med.*, 1917-18, xi., 289.

⁴ D. N. Paton and L. Findlay, *Quart. Journ. Exp. Phys.*, 1916, x., 318.

same reason equally good results could be obtained by the administration of a non-calcium salt, *e.g.*, ammonium chloride.¹

(*b*) *Alkalosis as a Cause of Tetany.*—In 1918 Howland and Marriott² described tetany following the administration of large doses of bicarbonate of soda to infants suffering from the acidosis accompanying gastro-enteritis, and although this is not such a frequent occurrence as these authors suggest, there is no doubt that it does happen, especially if there is some disease of the kidney interfering with the excretion of the alkali and thus tending to raise the alkalinity of the blood. In 1920 it was shown by Collip and Backhus³ and others that tetany could be induced by over-ventilation of the lungs from forced breathing. By this procedure excessive amounts of CO₂ are washed out and again the blood tends to become more alkaline. From the same cause tetany has been observed in consequence of hyperpnœa as a sequel to lethargic encephalitis, and occasionally it is observed in the state of alkalosis accompanying hypertrophic pyloric stenosis.

As in rickets and tetany there is a tendency to alkalosis, and since the induction of acidosis is the most rapidly acting therapeutic measure, the idea that in this change in the acid-base equilibrium of the blood, or rather of the tissue juices, is to be found the true exciting cause has been strongly supported. This is the view vigorously contended by Freudenberg and Gyorgy,⁴ who believe that an alkalotic state of the blood and tissue juices acts by interfering with the ionisation and physiological activity of calcium.

(*c*) *Parathyroid Disease and Guanidin Poisoning.*—That the disease depends on a toxic substance, either elaborated in the body from some metabolic error, or absorbed from the gut, is a view which has been held for a long time. The rarity with which spasmophilia occurred in the breast-fed infant, and the fact that cow's milk tended to make it worse, added some probability to such a theory. The protein, the fat, and the salts were all blamed in turn. The view expressed by Fuchs⁵ that occupation tetany was of the nature of ergot poisoning

¹ J. B. S. Haldane, *Journ. Phys.*, 1921, lv., 265.

² J. Howland and W. M'K. Marriott, *Quart. Journ. Med.*, 1917-18, xi., 289.

³ J. B. Collip and P. L. Backhus, *Amer. Journ. Phys.*, 1920, li., 568.

⁴ E. Freudenberg and P. Gyorgy, *Jahr. f. Kinderh.*, 1921, xcvi., 5.

⁵ A. Fuchs, *Wien. med. Wochensch.*, 1911, xli., 1854, 1919, 1974.

suggested to Biedl¹ that decomposition of histidin with the production of an amine simulating ergotoxin might be the exciting factor, but it was subsequently shown that this particular amino-acid was non-toxic.

Ever since the inception of the experimental study and surgery of the thyroid gland it has been recognised that carpopedal spasm and convulsions resulted from its removal. After Gley² in 1891 differentiated between the thyroid and the parathyroid glands, it was shown by Vassale and Generali³ in 1896 that the nervous symptoms which developed after thyroidectomy were in reality due to the removal at the same time of the parathyroid glands. The similarity between idiopathic tetany and the so-called parathyroid tetany had long been noted, and is now generally recognised. In fact, in 1902 it was suggested by Jeandelize,⁴ and again in 1905 by Yanase,⁵ that idiopathic tetany was due to disease of the parathyroids. The latter stated that he had found hæmorrhage into these structures in the spasmophilic infant, but his findings have not been verified by subsequent workers, and, if disease of the parathyroid gland does play any part in the etiology of idiopathic tetany, it is in response to changes of a functional rather than of an anatomical nature.

In 1909 MacCallum and Voegtlin⁶ described a fall in the blood calcium after parathyroidectomy, an observation which seemed not only to explain the relationship between these two types of tetany but also to lend support to the calcium depletion theory. The fact, however, that in parathyroid tetany, just as in idiopathic tetany, bleeding and perfusion with saline relieves the symptoms was a serious objection to the adoption of any depletion theory, and rather suggested that it was the presence of some added substance which was the exciting factor.

Various products of metabolism have been suggested as responsible, *e.g.*, ammonia, xanthin, β -iminazolyethylamine, and guanidin. The observations of Pekelharing⁷ on the relationship

¹ A. Biedl, *Innere Sekretion*, 1916, p. 125.

² E. Gley, *Compt. rend. soc. de biol.*, 1891, lxii., 841.

³ G. Vassale and F. Generali, *Arch. ital. de biol.*, 1896, xxv., 459.

⁴ Jeandelize, *Insuffisance thyroïdienne et parathyroïdienne*, Paris, 1903.

⁵ J. Yanase, *Jahrb. f. Kinderh.*, 1907, lxvii., 57.

⁶ W. G. MacCallum and C. Voegtlin, *Journ. Exp. Med.*, 1909, xi., 118.

⁷ C. A. Pekelharing, *Zeitsch. f. physiol. Chemie.*, 1911, lxxv., 207.

of methyl-guanidin acetic acid (creatin) to the tone of muscle, the absence of creatin from the urine of infants under six months of age (when tetany is rare), and its constant presence irrespective of diet in the urine of children between two and fifteen years (when tetany is not uncommon), as well as the fact that methyl-guanidin appears in the urine of dogs after parathyroidectomy (Koch),¹ suggested to Paton and Findlay² that this product of protein metabolism might be the causative factor. These workers not only confirmed Koch's observation that guanidin is present in the urine after parathyroidectomy, but that it could also be recovered from the blood, and that it was present in the urine of children suffering from idiopathic tetany. They further showed that in the cat, by the intramuscular injection of guanidin-hydrochloride, symptoms of tetany similar to those following parathyroidectomy could be induced, and they concluded that idiopathic tetany was due to dysfunction of the parathyroid gland by causing a perversion of protein metabolism and the production of a methyl-guanidin compound.

This view has not received general recognition, but strong support has been given to it by the recent work of Morris, Watson, and Morris³ on the metabolic changes found in guanidin poisoning. These workers have shown that in guanidin poisoning there results, just as in idiopathic tetany and after parathyroidectomy, a fall in blood calcium and a definite though slight alkalosis. The latter change is in consequence of a deflection of the acid radicle chlorine from the blood to the tissues, which they suggest is for the purposes of detoxication of the guanidin compound. In the light of this hypothesis one sees a probable explanation of the beneficial action of acidosis induced by the administration of calcium or ammonium chloride, as in this way a greater amount of the detoxicating element becomes available. When we recollect, too, that in rickets there is a disturbance of calcium metabolism, as well as a tendency to alkalosis, we can understand how these two diseases (rickets and tetany) should be so frequently associated. The very changes which favour the action of the toxic element (guanidin) are already in being and thus an amount of the toxin, which

¹ W. F. Koch, *Journ. Biol. Chem.*, 1912, xii., 313, and 1913, xv., 42.

² D. N. Paton and L. Findlay, *Quart. Journ. Exp. Phys.*, 1916, x., 377.

³ S. Morris, A. M. Watson, and N. Morris, *Biochem. Journ.*, 1931, xxv., 786.

could be dealt with easily by the healthy tissues, will call forth symptoms when rickets is present.

Treatment.—The method of treatment to be adopted depends on the nature of the tetany and the urgency of the symptoms. The latent stage of the disease, with occasional and mild attacks of laryngismus, is easily cured by the application of a thorough antirachitic regimen—fresh air, suitable diet, and the administration of an efficient cod-liver oil or radiated ergosterol. But when the attacks of laryngismus are frequent and severe, or when there are convulsions, more heroic measures are necessary because, so long as either laryngismus or convulsions are liable to occur, there is always the danger of a sudden and fatal issue. It is a wise precaution to leave such children with their mothers rather than admit them to hospital. The upset to the child in a hospital ward is a real danger. As John Thomson remarks, “a fatal attack of laryngismus has occurred in the course of a clinical demonstration.”

In the presence of convulsions the condition is most speedily controlled by an anæsthetic. This procedure, however, in no way acts as a cure but is simply a sedative, and on recovering from the anæsthesia the child is again liable to the convulsive seizures. As indicated while discussing the pathogenesis of the disease, active phenomena do not appear during the acidotic state, and to-day our most certain and rapid method of causing a disappearance of the active manifestations is to induce acidosis. This can readily be done by the administration of either calcium or ammonium chloride. The former drug is preferable because the ammonium salt is apt to cause toxic symptoms. In the milder cases fifteen to twenty grains of calcium chloride every four hours, and in the severe cases with frequent convulsions thirty grains every four hours, will usually cause a cessation of the active manifestations within a few hours, and a complete disappearance of all evidence of the disease within two or three days. But, like anæsthesia, acidosis is not a cure; it does not rectify the metabolic error causing the disease. It simply keeps the manifestations in abeyance. For a cure the administration of vitamin D, either in the form of natural or artificial sunlight, cod-liver oil, or radiated ergosterol, is essential. This therapy should be commenced at once, and by the end of a week will have brought about a normal metabolism, so that the administration of the acidosis-producing salt (calcium chloride) can be dispensed with.

CHAPTER XXIII

THE INTOXICATIONS

Acidosis and Alkalosis

DURING health the blood and tissue juices have a definite and slightly alkaline reaction which in disease may be departed from either in the direction of diminished alkalinity (acidosis) or of increased alkalinity (alkalosis). These changes in reaction are often accompanied by characteristic symptoms and even danger to life.

The change towards the acid side may result from a diminution of the alkaline elements (base) or, as is more frequently the case, from an increase in the acid elements (CO_2 , chlorine, the acetone bodies or protein katabolites) or a combination of these factors. The change to the alkaline side results from a diminution of the acid elements or an increase in the alkaline factors.

The various conditions under which these changes arise, the means of their recognition, as well as their significance, will be discussed in the following pages.

Acetonæmia.

The acetone bodies are perhaps the acid substances which are most frequently produced in excess in the body. Sometimes during the examination of a sick child our attention is attracted to their presence by a sweet chloroform-like smell of the breath. More frequently, however, it is from an examination of the urine that their presence is discovered. The appearance of acetone in the breath or in the urine (acetonuria) indicates acetonæmia and results from the abnormal production of bodies of the acetone series.

Significance of Acetonuria.

Acetonæmia may or may not have any clinical significance. The bodies of the acetone series or so-called ketones (acetone, acetoacetic acid, and β -oxybutyric acid) are intermediate

substances formed during the combustion of fat into CO_2 and H_2O . Normally, they are always being formed during the processes of metabolism, but they escape further disintegration to such a slight extent, and pass over into the urine in such minimal amounts, that they cannot be detected by ordinary clinical tests. Their detection clinically, either in the breath or in the urine, indicates that they are being produced in abnormal amounts, but this simply means that there is an incomplete combustion of fat—in short, that the normal metabolism is upset. Their presence has no reference to any particular diseased process.

This perversion of metabolism is a very common occurrence during illness in childhood, and as a rule has no more significance than a rise in the temperature. Fifty to sixty per cent. of children admitted to hospital present acetonuria of some degree and apparently quite irrespective of the nature and severity of the illness.^{1, 2} Both of these phenomena (fever and acetonuria) reveal that a state of health and perfect equilibrium have been departed from, but, and this cannot be too emphatically stated, acetonuria no more than the fever gives a clue to the nature of the diseased process. It may be that the excessive production of acetone bodies is due to a state of relative starvation from the abstention from food, or to an increased call for carbohydrate during the illness, to nervous or psychic influences, or to the normal activity of the cells of the body being upset in consequence of fever and toxæmia.

There is an unfortunate tendency to interpret the term acetonuria as synonymous with acidosis. Acetonuria, as we have pointed out, merely means that there is a state of acetonæmia due to an increased production of acetone bodies. Ketosis is the correct term for such a state. Ketosis, of course, may induce a condition of acidosis, but not necessarily so. In the vast majority of cases it does not cause acidosis. Indeed, acetonæmia with acetonuria may be present when there is a tendency to a more alkaline state of the blood (alkalosis). This is observed in the alkalotic states accompanying mountain sickness and hypertrophic pyloric stenosis. Acetonuria no more signifies acidosis than albuminuria indicates nephritis, or a cardiac murmur is evidence of *morbus cordis*. For a

¹ R. S. Frew, *Lancet*, 1911, ii., 1264.

² M. J. Brown and G. Anderson, *Arch. Dis. Child.*, 1926, i., 302.

diagnosis of acidosis there must be evidence of a change in the balance of the acid and base, or in the reaction of the blood, as, *e.g.*, is obtained from its CO_2 content, or is indicated by the distress of the body (hyperpnœa) in its efforts to keep the reaction of the blood and tissue juices within normal limits.

Acidosis.

Acidosis does not mean a definitely acid reaction of the blood. A true acid state of the blood never occurs. The circulation of a free acid, *i.e.*, one not combined with its equivalent amount of base, is, with the exception of carbonic acid, incompatible with life. Normally, one-twentieth of the carbonic acid is free, the rest being combined with base, and it is the ratio between the proportion which is free and that which is combined which is the ultimate factor in determining the reaction of the blood.

What is meant by acidosis is a disorder of metabolism which, if allowed to go on uncontrolled, would lead to an acid reaction, and the symptoms which we associate with the condition, as well as the tests which we apply for detecting its presence, are evidence of the means which the body adopts to hinder this from coming about. Acids, *e.g.*, carbonic acid, lactic acid, and phosphoric acid, are always being produced in the body as the result of katabolic activity, but in health their neutralisation and excretion are provided for through the activity of the blood and the excretory organs. So long as this production of acid remains within normal limits, and the organs engaged in their removal are healthy, the tissue juices and blood retain their normal reaction and there is no evidence of any disturbance. When, however, the production of acid is excessive in amount, or abnormal in variety, the efforts of the body must be increased if the normal reaction is to be retained. And, further, if the organs employed in the removal of these acids are diseased, not only will the difficulty in their elimination appear earlier but it will be proportionately greater. It is indeed only by some change in the functional activity of the excretory organs that one can explain the difference in the results of acidosis experimentally induced and that arising spontaneously. By the administration of a ketogenic diet (p. 1028), *i.e.*, one which contains an excessive amount of fat and an inadequate proportion of carbohydrate, there may result a degree of acidosis from the excessive production of ketones

which equals that of a most marked example of spontaneous acidosis, but symptoms are conspicuous by their absence. In these circumstances the body, being healthy, is able to cope with the acid tide; indeed, in spite of a continuation of the abnormal diet, the metabolism within a day or so accommodates itself so that the fat is more completely burned and the production of ketones steadily falls. Such an acidosis, because there are no symptoms, is called a "compensated acidosis."

It is thus seen that in retaining the blood and plasma fluids at the normal level the matter is one of balancing the loss with the production, and the parallel between acidosis and fever must occur to one's mind. Both acid and heat are produced under normal conditions and the abnormal state of fever or acidosis results from a disturbance of the balance between production and loss. Neither, however, can be called a clinical entity. Each (acidosis and fever) is merely evidence of a disordered action of the tissues, but neither is characteristic of any particular diseased process. Nevertheless, on account of limitation of knowledge, it may not be possible to discover the underlying pathological condition producing either acetonæmia or fever, and one may require to rest content with a diagnosis of acidosis or febricula. Such a diagnosis, however, should never be made unless as a last resort, and only after a thorough search for the root of the mischief has been made. But just as the tissues of the body have their optimum activity at a certain definite temperature, so also do they function best when the blood and tissue fluids have a certain definite reaction, and hence it is a wise therapeutic measure to reduce both to the normal level.

Alkalosis.

Alkalosis is the opposite of acidosis, being a state in which the blood and tissue juices are tending to become more alkaline than normal. It may result from an increased loss of acid, *e.g.*, CO₂, through over-ventilation of the lungs, or possibly of chlorine in the gastric juice in pyloric stenosis, or from a want of balance between absorption and excretion of base, as may occur during the administration of alkali to a patient with defective renal excretion. Thus we see that, just as is the case with acidosis, the functional state of the excretory organs is a factor of prime importance in its production.

As the normal reaction of the blood is alkaline, an increase in this direction would *a priori* seem to be less dangerous, and in practice such indeed is the case. Nevertheless, since the activity of the tissues has its optimum at a particular reaction, the body attempts to keep this at as near the normal level as possible, and the manifestations of the condition, and our evidence of its presence, are, just as in the case of acidosis, the means by which the body takes to do this.

Gaseous and Non-Gaseous Acidosis and Alkalosis.

Acidosis and alkalosis are conveniently divided into gaseous and non-gaseous types, depending on whether the factor inducing the change in reaction is primarily one affecting the CO_2 or some other acid or base. This is an important distinction because not only are the clinical manifestations of the particular change in reaction different in the two types, but the usual and most convenient laboratory evidences (total CO_2 content of the blood) are exactly the opposite. While a gaseous acidosis is accompanied by hypopnœa and a non-gaseous acidosis by hyperpnœa, a gaseous alkalosis will have as its most striking feature hyperpnœa and a non-gaseous alkalosis hypopnœa. In the one case the change in the respiration is the cause and in the other the effect. For example, opium poisoning will depress the breathing and cause a retention of CO_2 with a rise in the acidity, and conversely an increased rate or depth of breathing tends to increase the excretion of CO_2 with a resultant diminution in acidity. Thus the blood in gaseous acidosis shows a high CO_2 and in a non-gaseous type a low CO_2 , whereas in gaseous alkalosis the CO_2 is low and in non-gaseous alkalosis high.

There is, however, one clinical manifestation which, when present, does permit of a differentiation between the two states of acidosis and alkalosis. We have already (p. 550) drawn attention to the fact that the induction of acidosis is the best therapeutic measure in the treatment of tetany. For some reason not perfectly understood tetany and acidosis cannot co-exist. On the other hand, in alkalosis, no matter what the cause, and irrespective of the type, there results an increased mechanical and electrical excitability of the nervous system with the development of a facial phenomenon, carpo-pedal spasm, and convulsions.

Evidence of Acidosis and Alkalosis.

It has already been remarked that, with the exception of carbonic acid, no free acid can circulate in the blood but must be combined with base, and since the total acid and basic elements which go to form the reaction of the blood are perfectly balanced, and practically constant per unit volume, only a certain definite amount of acid can be carried. The presence of an abnormal acid, *e.g.*, ketones, or an increase of one normally present, *e.g.*, chlorine, as results from the administration of calcium chloride, must displace an equal amount of one of the others. This displacement usually affects carbonic acid because it is the most volatile of the acids in the blood. Thus a reduction in the proportion of the carbonic acid is the index of the tendency to an increase of the acid elements in the body fluids (acidosis), just as an increase in the proportion of carbonic acid indicates, as a rule, a tendency to the development of a more alkaline blood (alkalosis).¹ One might suggest that it is to aid the more rapid excretion of carbonic acid that there occurs in the acidotic state the characteristic symptom of hyperpnœa, just as its diminished excretion is favoured by the slow shallow breathing in alkalosis.

It must be remembered, as we have already pointed out, that a lowered carbonic acid content of the blood does not always mean a tendency to an increase in acidity. The reduction in the carbonic acid may be the primary change and result from over-ventilation of the lungs. This occurs from forced breathing, which may be performed voluntarily or arise spontaneously at high altitudes (mountain sickness), or in consequence of the hyperpnœa occasionally present in encephalitis lethargica. In these instances the loss of the normal acid of the blood is the primary change; the carbonic acid is, as it were, pulled out rather than pushed out, to make room for more acid, and hence there is the tendency to a

¹ The CO₂ content of the blood (venous) does not of course indicate the reaction of the blood, but it is the most convenient reliable method of arriving at this. The reaction can be estimated electrically, but this demands an elaborate apparatus. A simple colorimetric method has been devised by Dale and Evans, but it only indicates gross changes and does not reveal the body's method of controlling the change. The reaction of the blood may also be determined from the CO₂ content of alveolar air and the arterial blood, but neither of these methods is applicable in the child.

reduction in acidity—the state of so-called alkalosis. Nature, however, will attempt to keep the reaction at the normal level, and in this instance it results from an increase in one of the other acid elements, *e.g.*, chlorine, or by an increased production of ketone bodies. Here again we may draw attention to the fact that acetonuria is not evidence of acidosis. Nor does a high blood carbonic acid content always indicate alkalosis. If, as occurs in opium poisoning, the respiration is depressed the CO_2 accumulates and tends to a more acid reaction. It is thus apparent that neither the carbonic acid content of the blood nor the rate of the breathing, any more than the presence of acetone in the urine, enables one at any particular moment to say in which direction the reaction is tending. In order to decide whether the particular carbonic acid content of the blood spells acidosis or alkalosis, a knowledge of the conditions under which the abnormal state has developed—in short, the clinical history—is essential.

The above so-called buffer action of the blood is not the sole means of the body's protection against a change in reaction developing. The excretory function of the lungs and kidneys and of the intestines also plays an important part. Excessive amounts of CO_2 are got rid of by the lungs. This may be revealed by the characteristic hyperpnœa. The volatile acids of the oxybutyric series are also in part excreted by the lungs and thus there results another characteristic symptom, the smell of acetone in the breath. By the kidneys there is increased acid excretion, increase in ammonia formation to aid the excretion of acids and to neutralise tissue juices, increase in fixed base excretion, *e.g.*, calcium, from the bones and sodium and potassium from the tissues generally. As it is the salts of the body which in great part retain fluid their loss at the same time brings about an escape of fluid. These several points reveal how important is the health of the various excretory organs in preventing any change in the reaction of the blood and tissue fluids.

Both acidosis and alkalosis can arise, as already mentioned, under a variety of circumstances. Acidosis, for example, may develop spontaneously (cyclic vomiting); it may occur as a complication in the course of some diseased process (diabetes mellitus, gastro-enteritis, and nephritis); it may be the con-

sequence of the exhibition of certain drugs (salicylate of soda and calcium chloride) or the administration of an anæsthetic (chloroform). Alkalosis may develop during the course of hyperpnœa from the loss of carbonic acid, during the course of hypertrophic pyloric stenosis, perhaps from the loss of chlorine, or alkalosis may be the result of the administration of alkali when some disease interferes with its excretion.

Recurrent, Periodic, or Cyclic Vomiting—Periodic Acidosis.

This is a fairly well-defined clinical entity in which acidosis occurs apparently spontaneously at more or less definite intervals. Since vomiting is one of the most common, as well as most arresting manifestations of the condition, it is usually designated periodic or cyclic vomiting. This is, however, somewhat unfortunate, since the condition may occur without vomiting, and not infrequently pharyngitis, bronchitis, colitis, and enterospasm with colic may be the clinical evidence, and unless this is appreciated the condition will often pass unrecognised. A more satisfactory term would be *periodic* or *recurrent acidosis*, as this change in the reaction of the blood is invariably present and the periodic recurrence of the symptoms is one of the most striking features.

Of 48 cases vomiting was the chief symptom in 23, vomiting with diarrhœa in 8, bronchitis in 5, abdominal pain with or without fever in 4, and pharyngitis in 3. Fever during the attack was noted in 12 of the children, headache was complained of by 4, and in 2 of the patients convulsions had occurred during the attack.

The chief characteristic of the malady is its periodicity, recurring as it does with the greatest regularity in any individual case, but at different intervals in different patients. The attacks may for a time recur as often as every two weeks, but more often every one, two, or three months; sometimes they only occur once or twice a year.

The condition occurs, if anything, more frequently in girls than in boys: of the above-mentioned 48 cases 27 were girls and 21 boys. The children are commonly highly strung and nervous, and there may be a history of the same condition in one of the parents. It is a disease essentially of the non-hospital class. It is frequently stated that it is more common

in the only-child, but this would seem to depend on the fact that only-children in that same class are unduly common. In Fig. 176 are shown the relative size of the families in the acidotic cases along with the average relative size of the family in five numerically identical groups belonging to the same clientele.

The earliest age at which we have known the condition to commence is nine months, but the age of onset is usually between the second and third years. If untreated it persists throughout childhood but shows a tendency to spontaneous recovery about puberty, although it may continue or relapse in the form of migraine in later life.

As previously mentioned, the most frequent manifestation is vomiting. The child in apparently good health suddenly and without cause commences to vomit. On occasion it has been noticed that the child had been dull or irritable for a day or so previously, had "dark shadows" below the eyes, and that the breath had a strong odour. The vomiting varies in severity and duration; in some cases it increases in severity and frequency with great rapidity, so that within twelve hours not even sips of water can be retained; the vomitus consists of bile-stained fluid and in severe cases may contain a little blood.

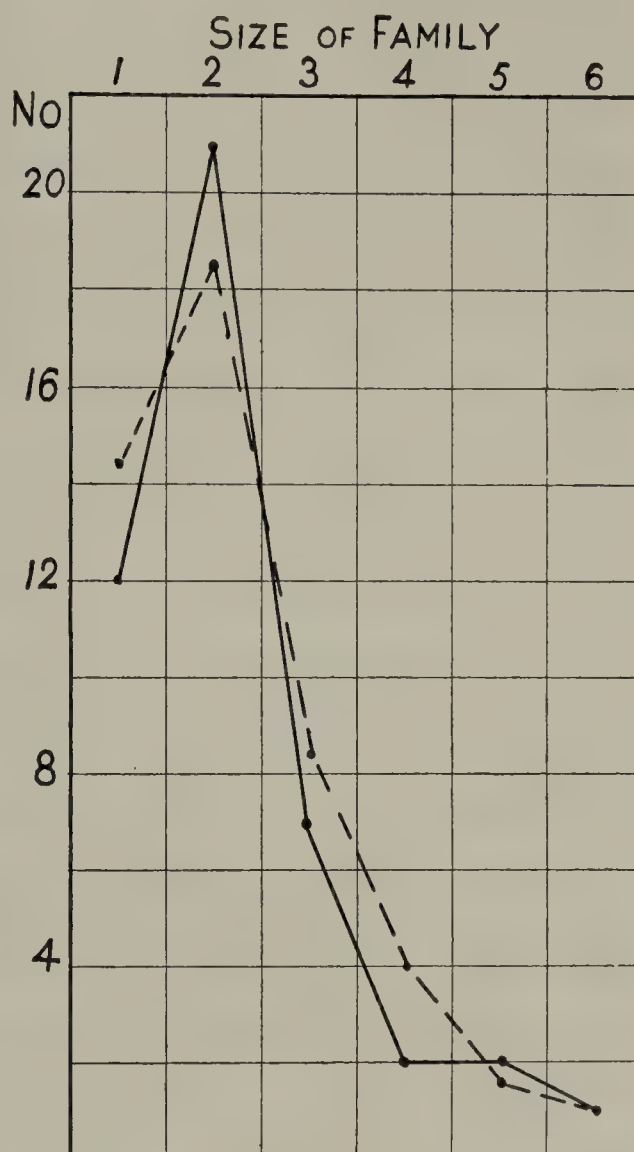


FIG. 176.—Showing size of family in Cyclical Vomiting.

— Cyclical vomiting.
 - - - Control families for same social class.

There may be complaint of headache and there may or may not be fever. The child is often restless and tosses about in bed; the breathing is deep and sighing (air hunger) and may be rapid, and the breath smells of acetone, which will be found in varying abundance in the urine. In the very severe cases the child becomes collapsed, with a small thready pulse, and at

this stage there may be some cyanosis. As a rule the bowels are constipated but at times there is diarrhœa.

Physical examination reveals nothing abnormal except the acetonuria and perhaps some abdominal tenderness, probably induced by the retching, but which, especially if there is fever, suggests appendicitis or the "acute abdomen," demanding immediate surgical intervention.

The collapse, with Hippocratic facies, small thready pulse, and extreme restlessness often seems to herald an early fatal termination, when the vomiting ceases as suddenly as it began, fluid nourishment is taken greedily and retained, and perhaps within twelve or twenty-four hours, but usually within two or three days, the child is out of all danger. He recuperates quickly and remains quite well until the next attack is due, in spite of all sorts of dietetic indiscretions and nervous excitement.

In some cases vomiting is a very minor feature of the illness and may, indeed, be absent. Fever and looseness of the bowels, attacks of abdominal pain, headache, bronchitis, and pharyngitis may be the manifestations.¹ In one child the typical vomiting attacks which had recurred for several years became replaced by attacks of bronchitis with an identical periodicity. In another child subject to typical attacks, severe intestinal hæmorrhage was the dominating feature in one of them; in this patient, owing to the uncertainty of the cause, abdominal section was performed, without, however, disclosing anything abnormal, and fortunately the child made an uneventful recovery.

Etiology. — The pathogenesis of cyclic vomiting is still obscure. It is certainly not due to auto-intoxication from the bowel. Nor is it due to an idiosyncrasy to any article of diet, as the child will go along in perfect health, eating anything and everything, until suddenly, as it were at the appointed time, *i.e.*, at the proper period, and without warning, the attack sets in. Nor does the attack occur from overstrain in an unduly nervous or highly-strung child, since it is only if the child attends a "party" or other exciting entertainment at the appropriate date that any etiological relationship can be discovered. Between the attacks the child can stand with impunity any

¹ Many of these cases have been described by Rochford (Starr's *Diseases of Children*, 2nd edition, London, 1898, p. 94) under the term "lithæmia," and by Stoelzner (*Oxyopathie*, Berlin, 1911) as "oxyopathie."

amount of so-called nervous strain, and is usually stated by his mother to be specially well.

It is a curious fact, too, that seldom does an attack supervene while the child is kept under observation either in hospital or in the out-patient department, even although no prophylactic measures are adopted. In only one instance out of many carefully-watched have we seen this happen.

The condition is not a mere matter of an arrest of the complete combustion of fat and the increased production of acetone bodies, since symptoms cannot be induced, even in a child who is subject to attacks of cyclic vomiting, by the administration of a ketogenic diet (fat-rich and carbohydrate-poor) so as to produce an abnormal amount of ketones. In one example, at the very onset of an attack, one of us (L. F.) deliberately put the child on such a diet with the intention of increasing the ketosis, but the child, nevertheless, made a speedy and uninterrupted recovery.

It has been suggested that it is a fall in the blood sugar and thus in carbohydrate available for use in the combustion of the fat which precipitates the attack, and that for some reason, perhaps as the result of a nerve crisis, this is more likely to occur in children subject to cyclic vomiting. In our experience there is no evidence in favour of such a theory. The blood sugar may be, but as a rule is not, unduly low during an attack of cyclic vomiting.

One cannot escape the conclusion that the condition is in great part nervous, but the *modus operandi* is at present impossible to define. In some way the regulating mechanism governing the balance between production and loss, or neutralisation of the ketones, is upset. As Morris demonstrated in one of our cases, the ammonia coefficient is low at the onset of an attack and rises as the patient improves, which is the reverse of what happens in an artificially-induced acidosis. This suggests a sudden disturbance of liver function, perhaps in consequence of a nervous phenomenon affecting the hepatic nerves, causing an inhibition of deamination and thus not providing the kidney with the necessary nitrogen for conversion to ammonia. In the same way impairment of liver function would interfere with oxidation and tend to hypoglycæmia. That impairment of liver function is present during the attack is revealed by the occurrence of a positive lævulose test.

Diagnosis.—As the condition is essentially one of acidosis, the diagnosis rests on the evidence of this condition: (*a*) clinical—hyperpnœa and nervous excitement or lethargy, and (*b*) laboratory—a low CO₂ content of the blood or other indication of a change in the reaction to the acid side. It may, however, be safely said that as a rule clinical evidence is sufficient. Since this variety of acidosis is due to a ketosis, *i.e.*, the increased production of acetone bodies, their presence in the breath or urine is invariable. But it must be borne in mind that acetone in the urine, or even in the breath, *per se*, is not grounds for a diagnosis. Acetonæmia with acetonuria is, as we have already remarked, a frequent occurrence in all varieties of diseased states during childhood.

In the first attack, and especially if vomiting is not the dominant feature of the illness, and more particularly if there is fever with delirium and headache, or some respiratory manifestation, the diagnosis may be extremely difficult. Meningitis, influenza or pneumonia may be simulated, but a careful physical examination will usually enable one to eliminate these conditions. It must also be remembered that acidosis, but as a rule without ketosis, may result from the administration of certain drugs (salicylate of soda and calcium chloride).

A history of one or several previous similar attacks is a most helpful diagnostic feature, and especially is this so when, as occurs not infrequently, the physician is consulted during the intervals between the attacks. In many cases, however, one can often only be certain of the nature of the condition, more particularly when the manifestations are anomalous, after watching the result of treatment. If the attacks have been truly acidotic in nature, then continuous administration of alkali will either cause their complete disappearance or a diminution in their frequency and severity. Should no amelioration result from such prophylactic therapy then the diagnosis must be revised.

Treatment.—When an attack begins, the patient must be kept in bed and the bowel emptied by a purgative, and, if that is vomited, by an enema. In most cases decided improvement follows the administration of bicarbonate of soda (120 to 180 grains per day) as first pointed out by Edsall.¹ Solutions of glucose or orange juice are also equally efficacious. The former neutralises the acid and the latter, from supplying carbohydrates,

¹ Edsall, *Amer. Journ. Med. Sci.*, 1903, cxxv., 629.

helps towards the more complete combustion of fat and a diminution in the amount of the ketones. On account of the tendency to vomit it is advisable to give the above remedies in small quantities and frequently.

If the vomiting is specially severe and defeats our therapy, this may be relieved by washing out the stomach with a solution of water and bicarbonate of soda. A poultice applied to the epigastrium is often helpful. Occasionally, if there is no suppression of urine, an injection of morphine may be of service. Saline and glucose enemata should also be given; they help by relieving the child's distressing thirst and encouraging the action of the kidneys, as well as in other ways. But the speediest method of controlling the acidotic state is the intravenous injection of a 10 per cent. solution of glucose. A solution of bicarbonate of soda would be equally efficacious, but it is difficult of sterilisation without causing its decomposition into the toxic carbonate.

It is, however, in the matter of *prophylaxis* that our therapy will meet with the greatest success. It is frequently recommended that the diet should be carefully regulated and that, for example, fat should be particularly avoided and, on the other hand, that carbohydrate be liberally supplied. In our opinion dieting has little effect on the course of the disease, except the regular supply of excessive amounts of glucose, but this line of treatment has the danger of upsetting digestion and of predisposing to caries of the teeth (p. 58). The fact that a ketogenic diet does not precipitate an attack, and that the condition will disappear while such a diet is being given, is surely proof that the dietetic factor is one of minor importance. It is, of course, unwise to allow the child to partake of excessive amounts of fat, *e.g.*, cream instead of milk, and the eating of green vegetables and fruit in moderation is advisable.

The best and simplest method of controlling cyclic vomiting is the administration between the attacks of bicarbonate of soda, given in doses of one-half drachm thrice daily after meals. This should be continued for at least a year or eighteen months, since, if it is omitted too soon, the attacks may return. If the periodic illnesses (bilious attacks, bronchitis, enterospasm, etc.) are definitely of the nature of an acidosis due to ketosis they will by this prophylactic therapy in all probability entirely cease. They will at least become less frequent and less severe.

Acidosis in Diabetes Mellitus.

Although, as we have said, acetonæmia with or without acidosis is an accompaniment of many diseased processes there are several maladies in which it is particularly liable to occur. The classical example is *diabetes mellitus* in which it must always be guarded against. This we do by keeping a careful watch on the urine for the appearance of acetone. The test for acetonuria in diabetes is comparable to the taking of the temperature in a case of tuberculosis. The treatment of this serious complication has already been discussed when dealing with the subject of diabetes (p. 536).

Acidosis in Gastro-Enteritis.

In fatal cases of *gastro-enteritis* a state of acidosis is not infrequently observed. This is in part due to the increased production of acid in the body from the necessary concomitant starvation, in consequence of the vomiting and defective absorption, apart altogether from any therapeutic deprivation of food, and in part to the loss of base which accompanies the loss of fluid by vomiting and diarrhœa. The acidosis is merely an indication of the severity of the lesion, and is in no way concerned with an explanation of the etiology of the illness. Nevertheless, it shows how the fatal termination may be contributed to, and it certainly suggests that the rational treatment is to replace the loss of fluids and salts and to hinder the production of acetone bodies by the administration of water, salts, and glucose. It may be stated, however, that the change in the reaction of the blood and tissue fluids can invariably be rectified by appropriate treatment, *i.e.*, the administration of alkalies, so that it may be taken as an axiom that a child with gastro-enteritis should not die from this complication.

Acidosis in Nephritis.

In *nephritis*, from the interference with the normal excretion of the acid products formed during the processes of metabolism, and probably also from the defective formation of ammonia, which helps to neutralise the waste products, a certain degree of acidosis may arise. It is usually present in the uræmic state, but in degree it is so moderate that it cannot be considered a really serious element in the case.

Post-Anæsthetic Acidosis (Delayed Chloroform Poisoning).

Rarely after anæsthesia a condition akin to acidosis develops. As it is believed to follow the use of chloroform more frequently than any other form of anæsthetic the condition is usually called "delayed chloroform poisoning." We have seen it occur after the use of ether and it has been recorded as a sequel to both local and spinal anæsthesia.

At varying periods (from a few hours to two or three days) after the child recovers from the anæsthetic there develop the usual symptoms of acidosis—vomiting, hyperpnœa without cyanosis, and extreme restlessness passing rapidly into coma and usually terminating fatally. There may or may not be acetonuria. The blood reveals the usual low CO_2 content and thus it would appear that the condition is one of simple acidosis, but, as is the case with gastro-enteritis, the administration of alkalies may cause all evidence of acidosis to disappear, yet the fatal issue is not prevented. Hence it must be concluded that there is more in the pathogenesis of the condition than acidosis. Pathologically there is a severe degree of fatty degeneration and even necrosis of the liver and kidneys. By some writers it has been suggested that fat embolism is the causal factor.

The important point about post-anæsthetic intoxication is that it should be prevented. This is attempted by avoiding intensive purgation and starvation previous to the giving of the anæsthetic, and that the preparation should consist in the administration of alkalies and glucose. This regime probably prevents its development in a certain number of cases, but it is by no means invariably successful. In the example referred to above, which followed the use of ether, the usual prophylactic treatment had been employed. In diabetes mellitus, of course, with insulin it is possible to employ a general anæsthetic with safety. This further suggests that acidosis is only a part of the picture in post-anæsthetic poisoning.

Salicylate Poisoning.

Salicylates if given alone to the child are extremely apt to cause acidosis. Even such moderate doses as 60 grains per day cause within two or three days a marked fall in the

carbonic acid content of the blood with definite symptoms of the acidotic state. Of these the commonest and earliest is vomiting, and if the drug be withdrawn at this stage a quick recovery is the rule. If, however, the drug is not discontinued hyperpnœa with fever, delirium, and coma develop, and a fatal termination may ensue.

The cause of salicylate poisoning has not been definitely determined. Acetonuria may or may not be present, but even when present it is slight in degree, so that ketosis is not the chief factor in its production. This is further borne out by the fact that glucose administered either prophylactically or therapeutically is of no value in this type of acidosis. Nor is the condition due directly to the presence of salicylate ions in the blood, since their amount bears no relationship to the severity of the acidotic state.

It has been shown by Morris and Graham¹ that the excretory function of the kidney plays an important rôle in its causation, so that this variety of acidosis bears an analogy to that met with in nephritis. These workers have demonstrated that during the administration of salicylates alone there is a diminished output of ammonia and a lessened power of the kidney to increase the concentration of the urine (urea concentration test). When the salicylate is combined with an equal or double amount of alkali (bicarbonate of soda) not only is there a greater amount of the drug present in the circulating blood, and a greater excretion by the urine, but the urinary output of nitrogen, chlorine, and water is higher. The practical lesson to be drawn from these findings is that salicylates should never be given alone, but always in combination with alkali.

Treatment.—In the presence of any of the manifestations of acidosis during the presentation of salicylates the drug should be withdrawn at once, a mild purge administered, and alkali (bicarbonate of soda) given in fairly abundant doses, *e.g.*, 10 to 30 grains every two or three hours. Vomiting should always be taken as a warning of impending acidosis. Even when the symptoms are severe (fever, delirium, and hyperpnœa) this treatment will cause their disappearance within twenty-four hours, and after an interval of a day or so the treatment can be recommenced. Although a recurrence of the condition is most

¹ N. Morris and S. Graham, *Arch. Dis. Child.*, 1931, vi., 273.

unlikely, it is wise to combine the drug with double the amount of bicarbonate of soda. In this way not only are toxic symptoms less likely to ensue, but the greater concentration of the salicylate ions in the blood, and their excretion in greater amount in the urine, suggests a quicker turnover and perhaps a greater efficiency of the drug.

Conditions Accompanied by Alkalosis.

Alkalosis, non-gaseous in type, is an almost constant accompaniment of *hypertrophic pyloric stenosis*. The etiological relationship between the pyloric obstruction and the alkalosis is fully discussed at p. 342. Alkalosis probably of similar etiology also follows high intestinal obstruction.

Many years ago MacAdam and Gordon¹ described an example of *periodic vomiting* in an adult of 39 years due to a non-gaseous alkalosis. The attacks recurred every three months or so, and were characterised by headache, lumbar pain, vomiting, and fever lasting for from four to seven days. There was no hyperpnœa, but acetone was constantly present in the urine during the attack. MacAdam found during the attacks a high CO₂ content of the blood as well as other evidence of a non-gaseous alkalosis from the examination of the alveolar air, and from the colorimetric estimation of the *pH* of the arterial blood. The final proof of this example of cyclic vomiting being due to alkalosis was the benefit obtained by the therapeutic and prophylactic administration of hydrochloric acid. We have never observed in a child cyclic vomiting due to alkalosis, and we know of no recorded instance in childhood.

As previously mentioned (p. 547), Howland and Marriott have described an alkalosis (non-gaseous in type), with the development of tetany, resulting from the *administration of large doses of bicarbonate of soda* to children when urinary excretion was impaired, *e.g.*, in gastro-enteritis. We ourselves have seen this to occur from the presentation of alkalies during the course of *nephritis*.

A gaseous alkalosis, as shown by Haldane and his co-workers, results in any circumstances in which there is a great washing-out of carbonic acid from the blood in consequence of over-ventilation of the lungs (increased respiration). Thus it may

¹ W. MacAdam and J. Gordon, *Lancet*, 1922, ii., 560.

be met with during the hyperpnœic sequel of encephalitis lethargica. Although we have seen a fairly large number of examples of hyperpnœa following epidemic encephalitis, we have only observed one child in whom alkalosis with tetany developed. It may be, of course, that symptomless (compensated) or milder degrees of alkalosis occur more frequently than the above experience would suggest, since it was the development of the tetany which directed attention to the alkalotic condition in this particular case.

CHAPTER XXIV

THE CARDIO-VASCULAR SYSTEM AND ITS DISEASES

THE most characteristic peculiarity of the child's circulation is its great readiness to react to all sorts of stimuli—physiological as well as pathological—by quickening, slowing, or irregularity of action, and by changes in the sounds and in the size of the heart. In childhood, therefore, alterations in the pulse, heart sounds, or dullness, such as might give cause for anxiety in an adult, are often due to mere passing phases of action which are of no practical importance. We may not be always able to explain how these changes are produced; but prolonged and careful clinical observation has shown that they do not imply any real weakening of the heart, and consequently that they call for no special treatment and especially for no restriction of the child's ordinary movements and habits.

Another point to be borne in mind in examining children's hearts is the relatively wide limits of the normal in the matter of such physical signs as the rate of the pulse, the position of the apex-beat, and the size of the heart as ascertained by percussion. These may vary considerably in different children, without there being any good reason for suspecting either threatened heart-failure or general ill-health.

The Pulse.

In the new-born child the pulse-rate usually varies from 120 to 140 in the minute. The following may be regarded as about the average rates in older children during sleep or perfect quiet:—

6 to 12 months	.	.	.	116 to 104.
2 „ 6 years	.	.	.	104 „ 90.
11 „ 14 years	.	.	.	84 „ 76.

The pulse may be quickened as much as twenty or thirty beats in the minute by mental emotion or bodily exercise. It is therefore of great importance to count its beats when the

child is quiet and not frightened, or, if possible, while he is sleeping, as well as after exercise or excitement.

Some of the characters of the pulse are well demonstrated by the sphygmograph; and good tracings may be taken from the radial artery even in young babies.¹ The best instrument to use is Dudgeon's, and it should be held in position by a soft

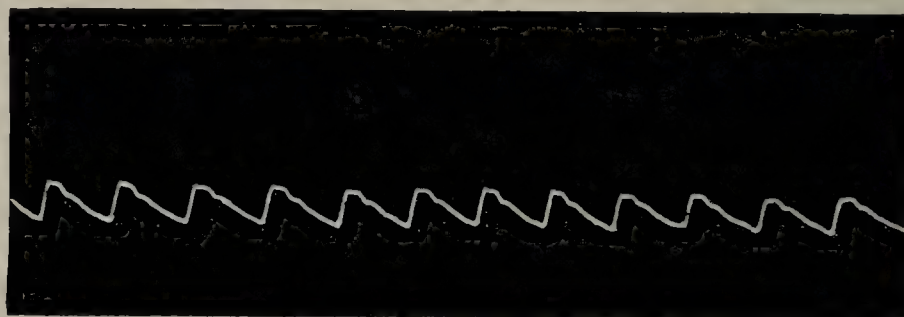


FIG. 177.—Pulse of normal infant of 6 days.

piece of elastic, instead of by the stiffer band often supplied by the makers.

In new-born children the pulse is very small, even for the size of the child, and the tracing shows the characters usually attributed to a relatively high arterial pressure. Its main peculiarities are that the up-stroke is somewhat slanting and that the dicrotic notch stands high on the down-stroke (Fig. 177). It recalls the tracing of aortic stenosis or that of some aneurism

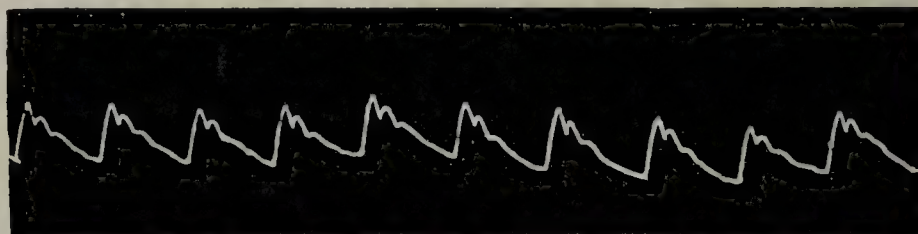


FIG. 178.—Pulse of child of 4 months.

cases. After the first few months of life the volume increases and a double summit develops (Fig. 178). The high-pressure type persists throughout the whole of the first year. At this

¹ See Dr H. O. Nicholson's articles on "The Pulse" in the *Encyclopædia Medica*; "On the Pulse in Infancy," *Scottish Med. and Surg. Journ.*, May 1901, 419; and "On the Pulse in Pneumonia in Children," in the *International Clinics*, 1902, 11th series, iv., 122. Dr Nicholson kindly gave permission to use the accompanying sphygmographs, which were all taken by him from patients of one of us (J. T.), and most of which have appeared in his *Encyclopædia Medica* article.

age dicrotism is rarely produced even by a very high temperature (Nicholson) (Fig. 179).

In children, the vagus is very sensitive to ordinary physiological stimulation, and normal irregularities of the pulse are therefore common. One example of this is the quickening of the pulse-rate during inspiration, with slowing during expira-

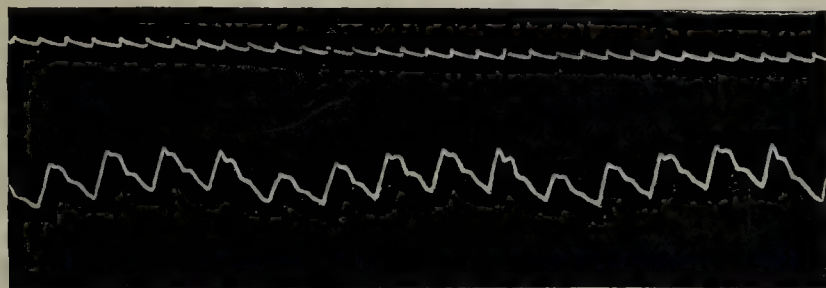


FIG. 179.—Pulse of child of 11 months. Temp. 105° F. No marked dicrotism.

tion, which occur when long slow breaths are taken. This effect of the respiration on the rate of the pulse is more distinct in children than in adults; and, according to Mackenzie,¹ its presence indicates a healthy state of the heart.

An allied phenomenon is the irregularity of the pulse which we frequently meet with in healthy children during sleep and also while awake, but also especially during recovery from any

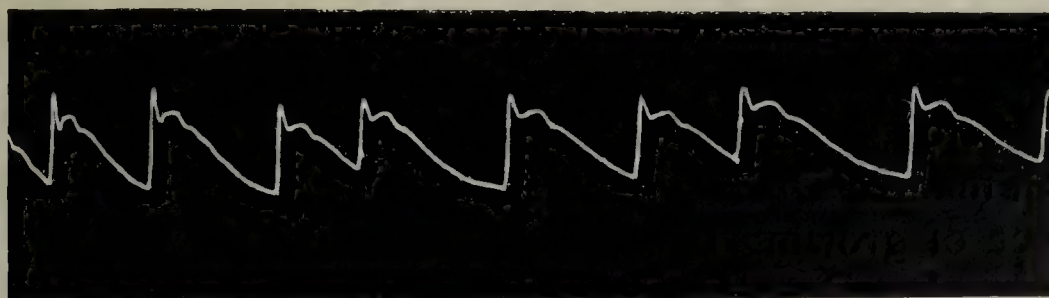


FIG. 180.—“Youthful type” of intermittent pulse in child of 2 years, with mild apical pneumonia. Did well.

feverish illness. This irregularity is always of the “youthful type,” that is to say, the diastolic periods of the cardiac cycle are of varying lengths, while the systolic periods remain of approximately equal duration (Fig. 180); and increased rapidity of the heart's action makes the pulse more regular because, the diastolic periods being shortened, their inequality becomes less noticeable. Some individuals, and some families, are

¹ *Principles of Diagnosis and Treatment in Heart Affections*, London, 1916, 143.

particularly prone to a high degree of this irregularity of the heart's action, but it is probable that it occurs in all children at one time or another; and its presence is to be regarded, not as a sign of disease, but actually as an indication that the heart-muscle is in a healthy condition. Its occurrence is certainly compatible with more than average athletic vigour.

The extra-systole or "adult type" of intermittency—in which the duration of the ventricular systole is variable (Fig. 181)—which is the common type in later life—is only occasionally met with in childhood. It may be met with in cases of heart-failure, but it is often compatible with apparent good health; but if there is a history of rheumatism one must be guarded in eliminating organic myocardial disease.¹

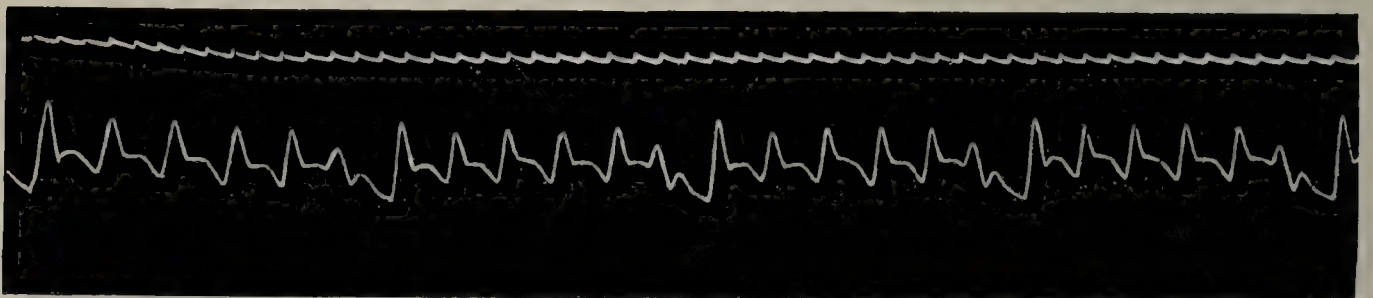


FIG. 181.—"Adult type" of irregularity in child of 5 years.

It is most important to recognise the harmlessness of the "youthful type" of pulse irregularity, because when this is not understood serious mistakes in prognosis and treatment are often made. During the last twenty years we have watched a number of growing boys and girls who for long periods had been treated as invalids because they showed a well-marked form of this irregularity, with or without functional cardiac murmurs, but without any other indication of disease in the heart or elsewhere. One used to be afraid of allowing these children to take the usual amount of exercise, believing that the irregular pulse must owe its presence to something which was a source of weakness, and that there was therefore a danger of heart-failure, unless special precautions were taken to limit the child's energies. The experience of years has gradually dispelled these anxieties, and proved that they were groundless. Some of the children referred to transgressed rules to a more

¹ T. Lewis, *Clinical Disorders of the Heart-Beat*, London, 1912, 53; L. Findlay, *Rheumatic Infection in Childhood*, London, 1931, 70 and 151.

or less considerable extent, yet none of them ever showed any sign of heart-failure after so doing; and in no single case did the heart symptoms develop into anything really serious. We are therefore prepared heartily to endorse Mackenzie's authoritative statement that "murmurs and irregularities may be manifestations of a perfectly healthy heart in the young."¹

In disease, as in health, the tendency to variability is a marked characteristic of the child's pulse. It is greatly quickened by fever, and when the temperature falls it becomes slow, and often at the same time irregular. In infants the pulse is not slowed by jaundice as it is in adults.

A very rapid pulse is often a help in the early diagnosis of scarlet fever; and in rheumatic cases it may indicate the onset of carditis. Generally, however, mere rapidity of the pulse has a much less serious significance in early than in later life.

Paroxysmal Tachycardia.—This obscure condition is very rare in children, but a few typical cases have been recorded.² Probably the youngest of these, a previously healthy boy of two and three-quarter years, was observed by R. Hutchison and J. Parkinson. He had four distinct attacks within as many months, with a pulse-rate between 215 and 245. When last seen he had kept well for four months.

The *prognosis* is generally serious, but it seems to depend on the duration of the attacks. Sometimes complete recovery takes place. Herringham's patient, for example, is reported by Sutherland to have been alive and working eighteen years after. If the attacks are severe, however, and last long, dilatation and heart-failure may occur and the case end fatally. In one case, a baby girl of seventeen months, the tachycardia was noticed on the fourth day of a slight general (? influenzal) catarrh—the pulse ranging from 208 to 215. Under treatment there was only slight temporary improvement, and the child died of heart-failure two days later with great dilatation, and with enlargement of the liver.

¹ "Some Manifestations of a Healthy Heart in the Young," *Brit. Med. Journ.*, 21st Dec. 1912, ii., 1697.

² Herringham, *Trans. Clin. Soc. Lond.*, 8th Jan. 1897, xxx., 99; Merklen, *Soc. Méd. des Hôp.*, 10 Mai 1901; R. Hutchison and John Parkinson, *Brit. Med. Journ.*, 1914, i., 534, and *Proc. Roy. Soc. Med.*, 1914, vol. vii. (Med. Sect., 117); G. A. Sutherland, *The Heart in Early Life*, 1914, 80.

The *treatment* of tachycardia by cardiac stimulants and sedatives is as unsatisfactory in children as in adults.

Pulse in Meningitis.—A slow and irregular pulse is frequently found in the early stages of tuberculous meningitis. Slowness and irregularity being, however, common in healthy childhood, this alone cannot be regarded as diagnostic. When

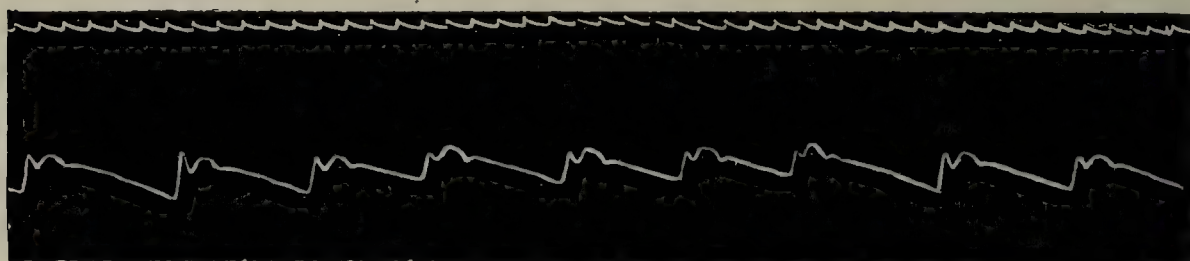


FIG. 182.—Pulse in Tuberculous Meningitis. (Child of $3\frac{1}{2}$ years.)
Radial artery contracted.

we also find the artery contracted so that it rolls under the finger, this is an important additional indication; and if the pulse remains slow and irregular when the temperature rises, this is also most significant of intracranial disease (Fig. 182). Under two years of age, marked slowing and irregularity of the pulse is less frequently found in tuberculous meningitis than it is in older children. In these young children the great irregularity of the respiration is even more characteristic than that of the pulse.

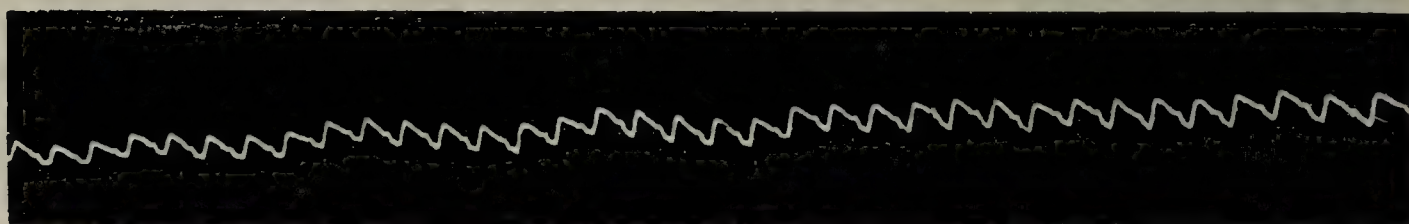


FIG. 183.—Very rapid pulse in last stage of Tuberculous Meningitis.
(Child of $2\frac{1}{2}$ years.)

In posterior basic meningitis the pulse is usually neither slow nor irregular, and shows no marked contraction of the artery. During the later stages of tuberculous meningitis in young children a very rapid pulse is often met with, the rate of which varies greatly in different quarter-minutes, although no distinct irregularity is detected by the finger alone (Fig. 183).

Pulse in Pneumonia.—The character of the pulse in pneumonia in children generally forms a far better guide

both to prognosis and treatment than do the physical signs in the lungs. Great lowering of the blood pressure is apt to occur, especially in broncho-pneumonia. When this happens in older children, and even in babies in bad cases, the pulse becomes dicrotic and finally monocrotic (Figs. 184 and 185).

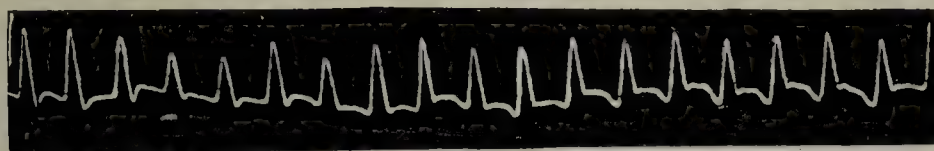


FIG. 184.—Pulse in Broncho-pneumonia ; of fatal significance if it persists for several days. (Child of 21 months.)

A rapid pulse with falling blood pressure is a matter of grave omen. Should the pressure fall so low that the pulse becomes monocrotic early in the case, or should this condition remain for several days, it is a very fatal sign.

Pulse in Nephritis.—Very high tension of the pulse in nephritis is rare in little children. It is more common in

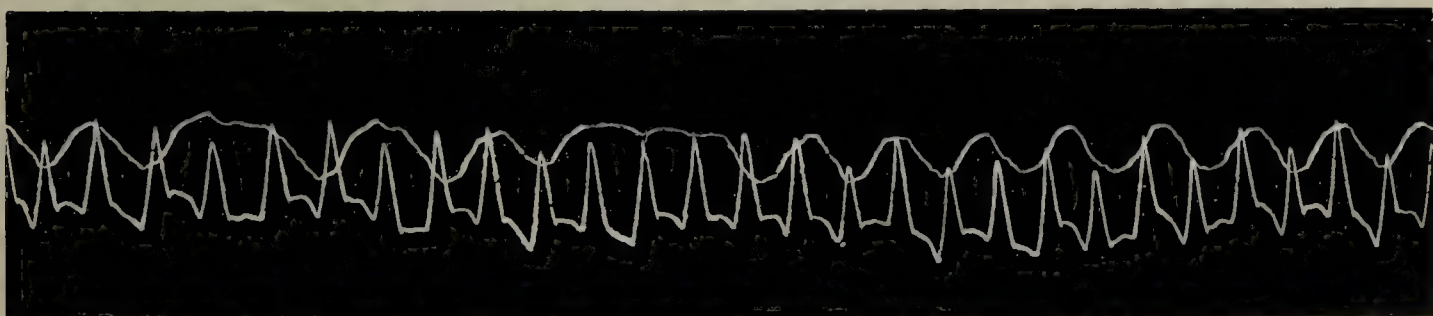


FIG. 185.—Monocrotic pulse from fatal Broncho-pneumonia. Pulse 160 ; respiration 76 ; temperature 104° F. (Child of 6½ years.)

them to find a fairly normal tension. A low tension pulse in nephritis is a bad sign in children as it is in adults.

Blood Pressure.

Observations on blood pressure are much less satisfactory in the child than in the adult, and one must be careful about drawing conclusions from isolated estimations. This is because there are so many variations even in health, and the results depend so much on the technique employed, and on the conditions under which the observations are made.¹

The best apparatus is the mercurial manometer, although an

¹ L. Findlay, *Quart. Med. Journ.*, 1911, iv., 489.

aneroid sphygmomanometer, if periodically tested, may be used. The reading varies inversely with the width of the armlet, being higher with a narrow armlet than with one of greater dimensions. For young children an armlet of two inches' width is considered suitable, and for older children, one with a width of three and a half inches. The auscultatory method of reading the pressure is to be recommended, since by palpation it is difficult in the child to be sure when the pulse is obliterated. Care must also be taken that the child is completely relaxed, as any straining causes a definite rise in pressure. The blood pressure is also temporarily raised after a meal, and last, but not least important, the blood pressure is proportionate to the height and weight of the child as well as to the circumference of the arm.

In the new-born the systolic pressure in the brachial artery averages only 45 to 55 mm. of mercury, but by the tenth day it has reached a height of 78 to 80 mm. During the first year of life it seldom exceeds 90 mm. and it remains at this level till the fifth year. At ten years of age the average reading is 100 to 110 mm., and at fifteen years 120 to 130 mm. The diastolic pressure is seldom, if ever, possible of estimation under seven years of age. At the age of seven years the diastolic pressure averages 65 mm., and at ten years, 70 to 75 mm. It is worthy of mention that it is seldom possible in the young child to estimate the blood pressure in the femoral artery.

Definitely pathological observations in children are met with comparatively rarely. In the vast majority of examples of acute nephritis the blood pressure is not raised. In the acute stage of the hæmorrhagic type there may be a moderate and temporary rise (130 to 140 mm.) for a few days. In chronic parenchymatous nephritis there is practically never any rise in the blood pressure. In chronic interstitial nephritis, on the other hand, a rise in blood pressure is frequently observed; but it is by no means the rule, and in that variety causing renal rickets a raised blood pressure is exceptional.

It must always be borne in mind that the pressure is proportionate to the height of the child, to the circumference of the arm, and to his weight, so that one must be careful in ascribing a high blood pressure in an obese child as evidence of endocrine mischief. Psychic influences must also always be considered. We have seen the systolic blood pressure of an

obese child in the out-patient department, and for a short time after admission to the ward, register 150 to 160 mm. fall after some days' residence to normal (115 mm.). It may be recalled that extraordinary high readings (150 to 180 mm.) have been observed in apparently healthy boys and girls just before and during the onset of puberty, but which later on fall to normal levels. It is probable that this temporary rise is due to the action of some internal secretion. Although, as a rule, these children suffer no discomfort, Abt and Feingold¹ record that some of their patients complained of nose-bleedings and a feeling of fullness in the head after exertion.

The Heart.

Whatever a child's complaints and symptoms may be, we can never safely omit a careful examination of his heart²; for some defects in that organ cause no subjective symptoms whatever, and need a local investigation to reveal them. The physical signs connected with the heart are only of importance in so far as they teach us something about the state of its structure, and especially about the way it is doing its work. We have to gather from them, not only whether the valves are competent, but whether the heart is normal in position, in shape, and in the size and strength of its different parts; also whether there is any pericardial effusion.

Inspection.—In cases of hypertrophy of the heart in children there is often considerable bulging of the soft chest-wall, and abnormal pulsations are more easily visible than in adults. *Venous pulsation* above the clavicle is common, especially when the children are lying, and it usually has no serious significance at all.

Palpation.—Palpation is relatively much more important in young children than in adults; and by its means cardiac hypertrophy and dilatation can usually be estimated more satisfactorily and accurately than by percussion.

The exact position of a normal apex-beat is often difficult to determine; and it may vary considerably in normal children, and also in the same child at different times and in different

¹ A. F. Abt and B. F. Feingold, *Kinderärztliche Praxis*, 1931, ii., 516.

² See Dr D. B. Lees' interesting address on "The Heart of the Child" in his work on *The Treatment of some Acute Visceral Inflammations, and Other Papers*, London, 1904, 252.

postures. In children under four years it may frequently be situated in or just outside the nipple-line, in the fourth intercostal space. This is because the heart lies more horizontally than in later life. As the child grows older the apex-beat is oftener found in the usual adult position. It may be displaced to one or other side by pleural effusions, or upward by pericardial effusion or extreme abdominal distension. In palpating we have to notice not only the position but also the degree of force of the apex-beat, and whether it is well defined, as in hypertrophy of the left ventricle, or diffuse as in enlargement of the right.

Distinct *pulsation* of the right ventricle in the *epigastrium* is always a most important sign. It means either displacement of the heart to the right, disease of the left ventricle, congenital heart disease, or, very often, that the right heart is dilating as the result of lung or kidney disease and is threatening to fail. The *liver-edge* should always be palpated at the same time as the apex, as hepatic enlargement from venous engorgement is a common and important accompaniment of heart-failure. *Thrills* are easily felt in children; and their character and distribution are important in diagnosis, especially in the case of congenital heart-disease.

Percussion.—Percussion is useful in childhood, though its results are often less definite and less easily interpreted than those of palpation, unless in the case of displacement. It is never necessary to percuss otherwise than lightly, or to use anything but the fingers.

To judge of the size of the *left ventricle* we percuss the heart's left border, which usually lies half a finger-breadth or so to the left of the apex-beat. When the left border is rapidly displaced outwards, so as to reach two or even three finger-breadths beyond the nipple-line, this indicates *dilatation of the left ventricle*. This diagnosis is confirmed by finding a feeble pulse, a feeble and diffuse apex-beat, a weak first sound at the apex, and accentuation of the pulmonary, and often also of the aortic second sound. Such dilatation indicates an urgent danger of syncope. It is to be watched for in such debilitating diseases as influenza and enteric fever, and especially in acute rheumatism and in diphtheria, both during and for some weeks after the throat affection. It is to be treated by keeping the patient lying flat for weeks.

The *right auricle* forms the right limit of the cardiac dullness and reaches from a half to one finger-breadth to the right of the sternum in the fourth space. When it is dilated, the dullness may extend two finger-breadths or even more from the sternum in this space, and from a half to one and a half in the third.

Great distension of the right auricle is an urgent danger-signal as it threatens death from asphyxia. It calls for immediate relief by means of leeches, venesection, or otherwise.

Displacement of the heart as evidenced by palpation and percussion may be due to transposition of the viscera, diaphragmatic hernia, pleural effusion, or fibrosis of the lung.

Auscultation.—The normal heart-sounds in little children are peculiar in certain ways. The first sound is often louder than the second in all the areas, so that even over the base of the heart the rhythm is trochaic and not iambic as in adults. The pulmonary second sound at the base is normally louder than the aortic. We have, therefore, to judge of the strength of the pulmonary second sound by comparing it with the first sound in the same area; and, only when the second is constantly the louder of the two sounds there, is it to be regarded as accentuated.¹ The heart must, however, be auscultated when the child is not frightened, because emotional disturbance normally causes a temporary accentuation of the pulmonary second sound.

Owing to the favourable conditions for conduction offered by the thorax in children, the heart-sounds are heard more widely over the lungs than in adults.

Functional Peculiarities of the Heart Signs.

Unusual physical signs, such as *murmurs* or an *increase in the heart's dullness with outward displacement of the apex-beat*, often indicate the presence of an acquired valvular disease or some congenital malformation; but this is by no means always the case. These signs, like the pulse irregularities already referred to, although unusual, are not necessarily abnormal; that is to say, they do not always indicate the presence of disease or even debility.

¹ C. Hochsinger, *Die Auscultation des kindlichen Herzens*, Wien, 1890, 13.

Functional murmurs are invariably systolic in time, and soft and blowing in character. They may be heard over the base or at the apex, but are commonest over the mid-sternum. Their loudness varies at different times and under different conditions; they are sometimes best heard when the patient has been resting, and sometimes after exertion; sometimes in the prone, and at others in the erect position. Their occurrence may be due to the presence of anæmia or to some dilatation of the heart. Often, however, we meet with well-marked murmurs when the blood is quite normal and no evidence of dilatation is present. These murmurs may also be noticed to come and go, without any corresponding changes in the size of the heart being demonstrable. The causation of these functional murmurs is not as yet understood; but it is certain that, in many cases, they are physiological phenomena which do not indicate the presence of anything that interferes with the heart's fitness for its ordinary work.

The question as to what extent an increase in the heart's dullness and an outward displacement of its apex may occur within the limits of perfect health, is one which cannot, as yet, be answered dogmatically. There can be no doubt, however, that small differences in these matters are of little moment, and that we should always be slow to lay much stress on even a considerable divergence from the usual, unless it is accompanied by obvious signs of hypertrophy of the heart muscle, persistent quickening and weakening of the pulse, or other indications of impaired function.

We have followed for years the cases of many children who had more or less well-marked functional murmurs, and have watched not a few of them through adolescence. In some there has been little change in the sounds to note; in others the character of the murmur has altered; in many it has entirely disappeared as the patient grew up.

In determining the question whether unusual physical signs do, or do not, indicate real disease or debility of the heart, our most important guide is the extent to which the heart is able to meet the strain of extra exertion without showing signs of failure. To ascertain this, we inquire as to the usual effect of exercise on the child; and we can sometimes ascertain it for ourselves by allowing the patient, if an older child, to run up stairs, or jump, or lift a weight, or, if a baby,

to scream and struggle, after we have made our examination. We then examine him again and note the effect of the exertions on his subjective sensations, breathing, colour, and pulse, and on the sounds and apex-beat of the heart.

Congenital Malformations of the Heart.¹

Causation.—Congenital malformations of the heart may represent an arrest in the growth and development of various normal parts of the organ, or an abnormal persistence of certain foetal structures which should have disappeared in the course of intra-uterine life. Until recent years many of the lesions found, such as thickening and puckering of the valves and adhesion of their cusps, were regarded as the result of intra-uterine endocarditis; but A. Keith² has shown that this was probably a mistake, and that these changes, like most of the congenital deformities met with in other parts of the body, are due to an obscure non-inflammatory process. The frequent co-existence of such malformations elsewhere, along with defects in the heart, is strongly in favour of this view.

We know very little, as yet, of the conditions under which arrests of development take place, but probably anything that interferes with the mother's health during the earlier stages of her pregnancy may favour their occurrence. One interesting fact, first pointed out by A. E. Garrod,³ is the curiously large proportion of cardiac, as well as other malformations which are found in cases of mongolism (p. 833). While it is probable that any form of debility in the mother may predispose to cardiac malformations in the child, there seems to be no good reason to regard either syphilis or rheumatism as of special importance in this connection. It is not very uncommon to find more than one case of congenital heart affection in the same family.

Clinical Phenomena.—The main symptoms and physical signs by which we recognise the presence of congenital malformation of the heart may be divided into three groups: (1) Cyanosis, which is usually accompanied by polycythæmia

¹ Maude E. Abbott, *Osler and M'Crae's System of Medicine*, 1908, iv., 323.

² "On Malformations of the Human Heart," *Lancet*, 1909, ii., 359, 433, and 519.

³ A. E. Garrod, "On the Association of Cardiac Malformations with other Congenital Defects," *St Bart. Hosp. Rep.*, 1894, xxx., 53.

and increase of the hæmoglobin in the blood, and often also by clubbing of the fingers and toes; (2) peculiar physical signs on inspection, palpation, percussion, and auscultation, which are generally quite different from those produced by the action of endocarditis on a previously normal heart; and (3) various indications of circulatory disturbance, such as general debility, infantilism, dyspnoea, and heart-pain; also, occasionally, recurrent epistaxis and epileptiform seizures of various types.

In several forms of malformation no symptoms at all may be present, or only cyanosis, a murmur, or debility. In some cases the chief clinical signs appear first about the time of puberty, when the rapid growth of the body is throwing an increased amount of work on the malformed heart.

I. **Cyanosis** is the most characteristic symptom of congenital lesions of the heart. Still found it present in about one-third of his cases. In 136 of our cases, 63 were cyanosed either at birth or later; while, in the remaining 73, no cyanosis was present on examination. This probably, however, overstates the real proportion of cyanosis, for cases of cardiac malformation without cyanosis are often overlooked, while those in whom it occurs are almost sure sooner or later to find their way to the physician.

The amount of discoloration varies greatly in different cases, and its degree is generally, though not always, an index of the severity of the case. It may be present from birth, or may only begin in the later stages of the case. Severe cyanosis never results in young children from ordinary endocarditis; and any cyanosis that occurs in that disease is always accompanied by indications of severe heart-failure.

The occurrence of cyanosis is probably due to a variety of factors. The deficient aeration of the blood, which may arise either from admixture of venous and arterial blood through abnormal openings, or from venous stasis, is probably the chief of these; but there can be little doubt that the great enlargement and new formation of the capillaries in the skin and elsewhere, and the deep tint of the blood, also play an important part in the causation of the cyanosis. Roger¹ states that cyanosis is only present when there is stenosis of the pulmonary artery and draws attention to the fact that when a

¹ H. Roger, *Bullet. de l'Académie de Médecine*, 1879, 2^{me} serie, viii., 1074.

patent interventricular septum is the sole lesion cyanosis is never observed.

In some children who are habitually cyanosed, and also not infrequently in others who ordinarily show no cyanosis, peculiar paroxysmal attacks of blueness sometimes occur. More or less severe seizures of this kind were observed in 10 per cent. of our cases of congenital heart lesion. They are usually accompanied by severe dyspnœa and pain of an extremely distressing character. They often set in as a result of dyspepsia and flatulence, and they may end in syncope or convulsions, or even in death. These attacks will be further referred to when we come to deal with heart pain.

Polycythæmia.—Along with cyanosis, there is usually an increase in the number of the red-blood corpuscles, and counts of from 6,000,000 to 9,000,000 are often found. This increase is apparently of the nature of a compensatory hypertrophy. The amount of the hæmoglobin in the blood is also usually proportionately increased. The number of leucocytes is seldom abnormal.

Clubbing of the fingers and toes occurs in most cases with cyanosis, but not in all. It may be present at birth, or may only develop after months or years, and is sometimes found without any cyanosis. A severe degree of clubbing never takes place in childhood from acquired heart lesions.

2. Physical Signs.—On inspection of the chest, visible pulsation in unusual areas is often observed, and alterations in the position and force of the apex-beat, and coarse thrills are frequently made out on palpation—especially at the base of the heart and over the right ventricle. In many cases changes in the size and contour of the heart can be demonstrated by palpation and percussion. On auscultation, alterations in the loudness of the sounds and murmurs, with unusual areas of audition and propagation, are commonly heard. The murmurs are nearly always systolic in time, but in a very few cases they are diastolic or presystolic.

The combinations of these phenomena are usually much more significant than their individual occurrence. It is often difficult or impossible to determine the exact area of maximum intensity of these murmurs, and this difficulty is, in doubtful cases, strongly in favour of the congenital nature of the heart lesion. The area of maximum intensity may also vary at

different times in the same case. If the patient becomes anæmic, the murmurs often alter considerably in character, and their areas of audition may become greatly enlarged. When a patient with a congenital heart lesion is dying in a state of great debility, the murmurs frequently diminish or even disappear.

3. Indications of Circulatory Disturbance.—Both in cases with cyanosis and in others there is sometimes great *debility*; they seldom thrive well during infancy and they are as a rule under weight and height for their age. *Dyspnœa* is common on exertion, though it may be absent when the child is at rest. It is chiefly seen in cases with cyanosis. Distressing spontaneous breathlessness usually accompanies attacks of paroxysmal angina.

Recurrent attacks of *heart pain with cyanosis*¹ are, as already mentioned, not at all uncommon. They usually begin gradually but sometimes become so severe that the child is quite stupefied and ceases for the time to notice what is going on around him. Sometimes he seems to lose consciousness altogether, and the seizure may end in a convulsion or even in death. When the patient has previously shown neither deep cyanosis nor a loud murmur, the occurrence of an attack of this kind may be the first thing to draw attention to the state of the heart.

Not a few children with congenital heart lesions are first brought to the physician on account of recurrent attacks of *epistaxis* or for *convulsions*.

The General Diagnosis of Congenital Heart Disease.—The lesions that may be found in these cases are not only very numerous, but they vary greatly in degree as well as in kind, so that their exact diagnosis is always difficult and indeed generally impossible. The difficulty is aggravated by the fact that it is far commoner to find several malformations present than one only, and in some instances the occurrence of secondary endocarditis may also further obscure the diagnosis. Fortunately, the prognosis and treatment of the individual case depends very little on the exact nature of the lesion present. They have to be decided by quite different considerations.

What is of real importance in the matter of a diagnosis is *first*, to settle whether the lesion is congenital or acquired,

¹ Variot et Sébilleau, *La Clinique Infantile*, 1 Mars 1904, t. ii., 137; and Sébilleau, "La Cyanose Congénitale Paroxystiques," *Thèse de Paris*, 1904.

which is usually not very difficult in young children; and *secondly*, if the condition is congenital, how much it is likely to interfere with the heart in the efficient performance of its functions. This is a matter of the greatest importance for both the prognosis and treatment.

It is generally easy in early childhood to distinguish the lesions of congenital from those of acquired heart disease. At this age acute endocarditis is rare, and when it does occur is mostly due to pneumococci or other pyogenic organisms, and secondary to empyema or acute epiphysitis. The murmurs which are most often mistaken for those of congenital heart disease are functional bruits of obscure origin, which are sometimes heard between the sternum and the left nipple-line, or over the base of the heart (p. 580). In a few cases such murmurs have to be watched for a considerable time before one can be certain that they are not due to a congenital defect. Generally, however, there is no difficulty, for these murmurs are never accompanied by cyanosis or clubbing, or by changes in the contour of the heart, and they are rarely loud or harsh in character.

The following **axioms**, taken mostly from Hochsinger's interesting work,¹ are sometimes helpful. They are founded on the observation of children under five years old.

1. *Loud, harsh musical murmurs with a normal or but slightly increased area of dullness* are not met with in little children except in congenital cases. When acquired heart affections occur at this age with loud murmurs, they invariably cause great increase in the cardiac dullness.

2. The occurrence of murmurs along with *greatly increased cardiac dullness* and a *feeble apex-beat* in young children is in favour of congenital disease. The increased dullness depends mainly on the right heart, while the left is but slightly altered. On the other hand, acquired heart disease in children is accompanied by increased force of the apex-beat, because its effect falls first on the left side, while the dilatation of the right heart sets in later and does not affect the strength of the apex-beat.

3. *Complete absence of murmurs at the apex*, while they are distinctly *present in the region of the auricles and over the*

¹ Hochsinger, "Ueber Diagnostik angeborener Herzfehler bei Kindern," *Wiener Klinik*, Feb. 1891.

pulmonary orifice, is always an important element in the differential diagnosis, and is more in favour of septal defects or congenital pulmonary stenosis than of endocarditis.

4. Abnormal weakness of the pulmonary second sound along with a distinct systolic murmur can, in early childhood, only be explained by assuming the presence of congenital pulmonary stenosis.

5. *Absence of a palpable thrill*, in spite of loud murmurs audible all over the præcordial region, occurs almost exclusively in cases of congenital septal defects, and is therefore against a diagnosis of acquired heart-disease.

6. *Loud systolic murmurs* (especially those accompanied by a thrill), which have their point of maximum intensity over the upper third of the sternum, and are unaccompanied by any sign of hypertrophy of the left ventricle, are important in the diagnosis of persistence of the ductus arteriosus, and cannot be explained by the assumption of endocarditis of the aortic valves.

7. A murmur in the second left interspace a short distance from the sternum, which begins after the first sound, lasts through the short pause and second sound into the long pause, and is accompanied by a corresponding thrill, is usually caused by a patent ductus arteriosus.¹ Such a murmur, however, is by no means always present in cases of this lesion.

8. The presence of congenital defects in other parts of the body is in favour of any heart affection which is present being of congenital origin.

General Prognosis.—In examining cases of congenital heart disease in early infancy little help as to the prognosis can be got from the murmurs present, as has already been mentioned. In most cases we have to be guided entirely by the child's general strength, his general development, the character of the pulse, and the presence or absence of cyanosis and enlargement of the heart and liver. Generally speaking, it is unfavourable in proportion to the degree of discoloration and of clubbing of the fingers present. In some serious cases, however, the cyanosis is usually slight or absent, but it recurs paroxysmally from time to time.

A large proportion of the cases of congenital heart lesion

¹ G. A. Gibson, "On Persistence of the Arterial Duct and its Diagnosis," *Edin. Med. Journ.*, July, 1900, viii., 1.

die within the first few weeks, and probably about two-thirds of them before the end of the second year. If the baby is well grown, well nourished, and vigorous, with little or no cyanosis and no increase of the cardiac dullness, an unfavourable prognosis as to his future should not be lightly given until he has been for some time under observation.

In older children the same points have to be considered ; but in most of the less severe cases the chief, and only satisfactory, way of estimating the patient's prospects of growing up and enjoying life like other children, and of judging how much or how little he should be allowed to do, is to inquire into the response of the heart to effort. To do this we have to find out what exertions he has been in the habit of making without dyspnœa or undue fatigue ; and we may try also how much he can now do without discomfort.

Should the child show any tendency to bronchial catarrh, this must be taken into consideration in the prognosis, for, in these children, that condition is very apt to pass into bronchopneumonia. For this reason also, measles and whooping-cough are especially dangerous in them. The tendency to the occurrence of ulcerative endocarditis has also to be remembered.

When a child with a congenital heart lesion is found to be suffering severely from general debility, it is always important, before giving a grave prognosis, to make sure that the symptoms are due to the state of the heart and not to other causes. We have, on several occasions, seen children who had a congenital cardiac malformation and were supposed to be dying from it, but whose alarming symptoms were entirely due to other causes, such as infantile scurvy, severe rickets, or some other food disorder, and who rapidly recovered strength under appropriate dieting.

The Diagnosis and Prognosis of Particular Lesions.—Although it is not necessary here to deal at length with the clinical manifestations of the numerous varieties of congenital heart lesions, a word or two may be added about the symptoms and prognosis of a few of the commoner types.

Congenital pulmonary stenosis is probably the commonest cardiac malformation met with in general practice, for Peacock¹ asserts that it is present in more than four-fifths of the children

¹ T. B. Peacock, *On Malformations of the Human Heart*, 2nd ed., London, 1866.

with congenital heart affection who survive to the age of twelve years. The patients are deeply cyanosed and generally have a loud, harsh pulmonary murmur which is often accompanied by a thrill. In some cases the pulmonary second sound is entirely absent.

Transposition of the large vessels is not very rare in young infants. In this condition the cyanosis may only be slight, but the patients are always very feeble and they usually die before the end of the first year.

Patency of the ductus arteriosus is one of the commoner malformations, but it is not very often the only lesion. When it does occur alone the patient generally enjoys good health for many years, and only begins to show cyanosis or to suffer from other cardiac symptoms in later life, if at all. The characters of the clinical phenomena probably vary a great deal according to the size and shape of the patent duct.

Patency of the foramen ovale gives rise to no symptoms or physical signs unless the opening is very large. Small apertures in it are scarcely to be reckoned as abnormalities, as they do no harm and may even in some circumstances have a favourable effect on the circulation. When the opening is large, however, it may occasion considerable interference with the action of the heart. The question as to the physical signs which it may produce is a matter of great difference of opinion. Its presence can never be diagnosed with certainty during life unless when severe cyanosis develops during lung or bronchial affections (*cyanose tardive*).¹

Defects in the upper part of the interventricular septum are common. They are occasionally the only defect present, but generally they are accompanied by other malformations. An opening in this situation, when it is the only lesion, may give rise to neither symptoms nor signs; but in most cases there is a loud harsh murmur over the third and fourth left costal cartilages, during the whole of systole and diastole (*murmur de Roger*),² and sometimes enlargement of the right heart. Cyanosis is noted in about one-half of the cases and a thrill in about one-third. Implication of the A.V. bundle may cause heart-block.^{3, 4}

¹ M. Hardy, *Gazette des Hopitaux*, 1876, xlvii., 665.

² H. Roger, *Bullet. de l'Académie de Médecine*, 1879, 2^{me} serie, viii., 1074.

³ G. B. Fleming and M. Stevenson, *Arch. Dis. Child.*, 1928, iii., 221.

⁴ J. G. Wilson and R. T. Grant, *Heart*, 1926, xii., 295.

As the foetal opening in the interventricular septum normally closes early in the eighth week of intra-uterine life, its persistence may be held to indicate a very early disturbance of the child's circulation.

Coarctation of the Aorta, associated with and without some cardiac anomaly, requires special mention, as it gives rise to a very definite clinical picture which enables a diagnosis to be established during life. Two varieties of the lesion are described: (*a*) that in which there is a diffuse narrowing of the arch of the aorta proximal to the ductus arteriosus, and usually associated with gross cardiac malformations, and (*b*) that in which there is an abrupt and localised constriction in the neighbourhood of the ductus, occasionally slightly above, at other times at the same level, but most frequently just below its insertion. The subjects of the former type of deformity seldom survive long after birth, and hence this variety is called the *infantile type of coarctation* and is looked upon as a true congenital malformation.

The abrupt type of constriction is much more seldom associated with other cardiac defects, and is compatible with a long life devoid of any circulatory disturbance. It is most frequently met with in the adult, in fact it has never been observed in the foetus, and is hence spoken of as the *adult type of coarctation*. Skoda¹ doubts if it really is a congenital lesion, and holds that it develops post-natally, probably as the result of an extension of the ductus arteriosus tissue into the surrounding aortic wall, and by its natural contraction brings about a gradual constriction of the aorta. This explanation is in keeping with the life-history of the condition. Symptoms and signs, especially the development of a collateral circulation, are as a rule late in appearing.

As noted above, there may be no symptoms, and not infrequently the condition is discovered accidentally during a routine examination or in the post-mortem room. The characteristic and pathognomonic features of the lesion, which are dependent on the obstruction to the circulation are (*a*) a very weak or impalpable femoral pulse, and (*b*) the development of a collateral circulation between the aorta above and below the constriction. This latter usually affects the internal mammary artery and its branches and the superior intercostals,

¹ Skoda, quoted by Maude E. Abbott, Osler, and M'Rae, *Modern Medicine*, London, 1927, iv., 774.

which may be felt pulsating in the interscapular spaces. Other symptoms present may be undue pulsation of the vessels of the neck and a feeling of fullness in the head, enlargement of the heart, a systolic thrill, and a systolic murmur which may be unduly wide in its distribution. In not a few cases the condition has been disclosed through the development of ulcerative aortitis at the seat of the lesion.¹

Treatment.—Many children with congenital heart lesions have to be treated as invalids all their life; and they may be made happier and more comfortable if their habits are carefully regulated so as to prevent unnecessary excitement and exertion and all causes of chill and of indigestion. They should always rest in the middle of the day, and their slightest ailments must be carefully attended to on their first appearance. School life is generally, though not always, out of the question. When anginal attacks occur, carminatives should be given after meals; and inhalation of amyl nitrite when the pain begins. If these measures fail to relieve the distress, it is usually necessary to have recourse to opium in some form. In ordinary cases of congenital heart affection, digitalis has no effect on the cyanosis and other symptoms, but should heart failure, epistaxis, or convulsions set in, it may be very useful.

In those cases with loud congenital heart murmurs, in which there is no abnormal fatigue or discomfort on exertion, no treatment is required. These children are practically in good health, and they should certainly be allowed to run about freely and exert themselves in any reasonable way as much as they like. Anæsthetics and surgical operations are no more dangerous for them than for other children.

Acquired Organic Heart Disease.

Simple Endocarditis may occur at any age; but it is rare in children under three. It may affect any of the valves, but in the great majority of cases it is the mitral which suffers. Aortic stenosis is especially rare in childhood, and cases with a basal systolic murmur, which have been diagnosed as due to this condition, not very rarely prove to be due to some less serious cause. It is characteristic of childhood that the myocardium and pericardium are often and severely affected.

¹ J. F. Poynton and W. P. H. Sheldon, *Arch. Dis. Child.*, 1928, iii., 191.

Symptoms.—The physical signs of endocarditis in children do not differ essentially from those in later life, but hypertrophy and dilatation occur more readily and more rapidly. The general symptoms are usually very indefinite. There may be a slight rise of temperature and pulse along with pallor, a short dry cough, breathlessness on exertion, palpitation, and emaciation. Even in severe cases of valvular disease it is rare in childhood to meet with the great œdema of the limbs, enlargement of the liver and spleen, dyspnœa, and cyanosis which are so commonly seen in the adult in advanced cardiac disease.

Causes.—Heart disease in children may arise from many causes, some of which are obscure in origin. The great majority of cases, however, are due to rheumatism, and the main thing we want to know about the cause of any case is whether it is or is not rheumatic. This question is important, because, if the lesion is rheumatic, it throws light on the child's tendency to disease and indicates much with regard to his present and future treatment. The absolute diagnosis of rheumatism must depend on the presence of one or more other manifestations of that disease in the patient, or on the past history of their having been present. If characteristic acute arthritis of one or more joints exists, that is usually held to be sufficient to settle the question; and the presence of erythema circinatum or of choreic movements has the same significance. The most satisfactory proof, however, of the presence of rheumatism is the finding of rheumatic nodules.

Treatment.—The treatment of a recent case of acquired heart disease in childhood differs in no important respect from that in adults. The first and main point, the importance of which it would be difficult to exaggerate, is that the child be kept lying, and all exertion avoided both during, and for weeks or months after, its onset. The period during which complete rest is necessary varies, of course, in different cases. The indications that the amount of rest may be lessened are slowing of the pulse, the appearance of the juvenile form of irregularity, and decrease in the size of the heart.

The next point is that if the case is, or may be, rheumatic, it should be treated actively by anti-rheumatic measures, including salicylates in large doses. Digitalis and allied remedies are valuable in suitable cases, but they must not be given indiscriminately, merely because the heart is affected, but

because there are signs of failing circulation. G. A. Sutherland¹ has recently shown that great advantage frequently follows the free administration of digitalin, along with anti-rheumatic treatment, in cases of rapid pulse in rheumatic children as well as in auricular fibrillation. In severe cases of chronic valvular disease in children, with heart-failure beginning, a course of grey powder may greatly assist the action of digitalis, just as blue pills often do in the case of adult patients.² In valvular heart disease due to rheumatism it is important to be always on the outlook for possibly rheumatic symptoms such as joint pains and sore throat, and to abort them by early treatment. It is also desirable that any source of recurrent infection, such as an unhealthy naso-pharynx or tonsils, should be put right. Digitalis is contra-indicated in diphtheritic heart lesions.³

Malignant, Ulcerative or Bacterial Endocarditis is decidedly rare during childhood. This is somewhat remarkable in view of the facts (1) that childhood is the age *par excellence* for septicæmia, and (2) that rheumatic heart disease, which is usually considered the most important predisposing factor, is so common in the early years of life. During the course of sixteen years one of us (L. F.) has only met with 8 examples of malignant endocarditis as against 473 of the rheumatic variety. It is worthy of mention, too, that in not one of them was there a history of rheumatism or any evidence of a congenital cardiac defect. There are, however, in the literature many examples of ulcerative endocarditis (during both childhood and adult life) supervening on some congenital cardiac lesion. One variety of malignant endocarditis, the so-called vegetative type due to the pneumococcus, may arise as a sequela of pneumonia.

Rost and Fischer,⁴ who have recently reviewed the literature on the subject, found that, of 63 cases under fourteen years of age, in not one instance had it been observed under five years of age. Of 12 cases under their own care all were between six and thirteen years old. Of our own 8 cases, with the exception of one child of fourteen months who presented post-mortem

¹ *Quart. Journ. Med.*, April 1919, xii., No. 47, 183.

² Wm. Murray, *Rough Notes on Remedies*, London, 1899, 3rd ed., 49.

³ Hugh M'Culloch, *Southern Med. Journ.*, Alabama, U.S., Feb. 1921, xiv., 110.

⁴ W. L. Rost and A. E. Fischer, *Amer. Journ. Dis. Child.*, 1928, xxxvi., 1144.

an extensive pyonephrosis and pneumonia at the right apex, in addition to ulcerative endocarditis of the mitral valve, the patients were all over five years of age. Dible¹ records an example of ulcerative endocarditis due to the streptococcus in a child aged six months who had shortly before suffered from impetigo. Occasionally the condition has been observed in the new-born, either due to a septic condition in the mother during pregnancy² or to sepsis developing in the child after birth.³

Malignant endocarditis rather than the simple (rheumatic) variety is always to be suspected in the absence of any history of rheumatism, in the presence or recent history of a septic focus, when the aortic valves are involved and when the fever is markedly remittent or intermittent (septic) in type. The occurrence of embolism, petechial eruptions, enlargement of the spleen, a marked leucocytosis, the presence of tender nodules on the hands, fingers, and toes (Osler nodes), and frequent changes in the auscultatory phenomena are characteristic of this variety of endocarditis. The finding of a pyogenic organism in the blood would be positive proof, but unfortunately this evidence is only obtained in a proportion of the cases.

The course of this variety of endocarditis is very varied; a fatal termination is seldom delayed longer than a year, but it must be remembered that a large proportion of examples of rheumatic endocarditis also terminate fatally within one year of the onset. Of 154 fatal examples of rheumatic endocarditis under the care of one of us (L. F.), 37.4 per cent. died within one year of the onset of the infection (p. 910).

Pericarditis.—In children under three years pericarditis is generally met with as a complication of empyema or pneumonia. Such cases of pneumococcal pericarditis are apt to be overlooked, because there are often no ascertainable subjective symptoms and also no friction. They are probably always fatal.

In older children the condition occurs most frequently as a manifestation of rheumatism. When this is so, it generally—although not always—runs a rather subacute course with little or no fluid effusion. If the case is at all severe the myocardium

¹ J. H. Dible, *Journ. Path. and Bact.*, 1920, xxiii., 196.

² S. S. Adams, *Trans. Amer. Pæd. Soc.*, 1902, xiv., 160.

³ A. von Reuss, *Die Krankheiten des Neugeborenen*, Berlin, 1914, 291.

is usually more or less extensively implicated, and dilatation of the heart occurs. Pericarditis is generally met with in children who have already suffered from other rheumatic manifestations (p. 903).

Occasionally severe pericarditis sets in suddenly in older children, apart from rheumatism, as a primary disease. In these cases the friction is very loud, but there are scarcely any other distinctive symptoms—merely a slight præcordial uneasiness with a rise of temperature. Complete recovery usually takes place if the case is recognised early and the patient kept in bed.

Pericarditis is not an uncommon occurrence in the course of scarlet fever and some other infective conditions; and it is occasionally met with in tuberculosis.

Treatment.—In slight cases of rheumatic pericarditis nothing beyond complete rest and general anti-rheumatic treatment is called for. When the case is at all severe, however, local measures are important.¹ The main danger in such cases arises from the tendency of the right auricle to become over-distended. It must therefore be closely watched by percussion in the fourth right intercostal space, and if it is dilating, leeches (four to six) should be at once applied over the heart or venesection done. An ice-bag should then be applied over the præcordial area, the patient's lower limbs having first been thoroughly warmed by hot-water bottles. The child should lie on his back, and the ice-bag should be placed over the heart, with nothing between it and the skin of the præcordia. If there is much local hyperæsthesia, the ice-bag should, to begin with, be suspended over the patient so that it just touches his chest. It should be refilled every hour and a half, and the hot bottles every three hours. Children usually like this mode of treatment and ask for it to be reapplied when it is to be discontinued.

Along with the local treatment, salicylate and bicarbonate of soda should, in most cases, be given internally.

Paracentesis pericardii is very rarely required.

¹ Lees, *Treatment of some Acute Visceral Inflammations*, London, 1904, 32.

Aortic Disease.

Disease of the aortic valves only is not common in childhood ; it is, however, observed in about 50 per cent. of the fatal cases of rheumatic endocarditis.¹ Aortic disease may also arise from congenital syphilis. Anginal pain due to aortic disease is rare.

Thoracic aneurisms are very rare ; but a number of cases have been placed on record.² The majority of these have been due to atheromatous changes in the vessel wall which have generally been attributable to congenital syphilis. A few, including that of Bronson and Sutherland, have occurred as the result of coarctation of the aorta. In exceptional instances an aneurism may arise from traumatism, from septic infection, or from embolism ; and false aneurisms may occur as the result of erosion from without.

Neurotic Heart Affection.

Severe cases of neurotic disturbance of the heart's action are occasionally met with in children of school age apart from any organic lesion. In these, there is a constant tendency to great rapidity of the heart's action with palpitation, on the least emotional excitement. Functional murmurs, with some dilatation, are often also present. Muscular exertion apart from excitement often causes no abnormal quickening of the pulse in these children, though the least emotional excitement does so at once. *During sleep the pulse rate is normal.*

These cases are very troublesome. They may last for years, and interfere greatly with school-work and with open-air games of all kinds.

The *treatment* is that of the general nervous state. It includes the avoidance of all causes of worry or excitement of any kind, much rest and sleep, and a routine of quiet occupation in familiar surroundings. The patient should be much in the open air. Digitalis is of no value ; but the prolonged administration of bromides is distinctly beneficial.

In cases that are carefully looked after the *prognosis* as to ultimate recovery is good. After the period of puberty, if not before, the nervous irritability gradually subsides, and in time the heart symptoms entirely disappear.

¹ L. Findlay, *Rheumatic Infection in Childhood*, 1931, p. 74.

² Edith Bronson and G. A. Sutherland, "Ruptured Aneurisms in Childhood," *Brit. Journ. Dis. Child.*, 1918, xv., 241.

Raynaud's Disease.

This name is given to a group of cases in which there occur, from time to time, paroxysmal attacks of extreme numbness and chilliness of the extremities, with a more or less symmetrical distribution. According to Raynaud, the local condition depends on an undue irritability of the vasomotor centre or centres, owing to which ordinary causes of stimulation, such as slight cold or a full meal, produce exaggerated effects and result in prolonged paroxysmal contraction of the peripheral arterioles. A proportion of the patients are the subjects of congenital syphilis.¹

Symptoms.—Raynaud describes the effects of the arterial spasm as occurring in three degrees of severity—local syncope, local asphyxia, and local symmetrical gangrene.

Local Syncope.—In this, one or more digits on each hand or foot become white and “dead.” The condition varies greatly in severity, being sometimes merely a slight exaggeration of the chilliness of the extremities which is natural to many children. There may be little or no pain, merely a degree of discomfort with some analgesia and blunting of the tactile sense. After the attack has lasted a few minutes—or a few hours—it passes off with a sensation of burning heat, and the affected part soon resumes its usual colour. The patient's general health seems unaffected. The attacks generally occur in cold weather.

Local Asphyxia.—In this the affected extremities, or digits, assume a deep dusky purple colour and become painful and tender. Generally the hands and feet are affected, less commonly the ears, and rarely the nose. Local asphyxia may succeed to local syncope, or it may occur without any previous pallor of the parts being noticed.

Local Symmetrical Gangrene.—This is the terminal stage of the two other degrees; fortunately, it is not often reached. Sometimes the extremities of the digits become gradually black and mummified. In other cases the necrotic process begins with the formation of bullæ on the surface of the cyanosed parts, as happened in the case shown in Fig. 186.

In a few cases of Raynaud's disease we meet with hæmoglobinuria at the time of the attacks. Mental torpor may be

¹ M. Vargas, *La Medicina de los Niños*, 1930, xxxi., 257.

present; and epileptic fits occasionally occur. Peripheral neuritis has been found in the affected limbs.

Diagnosis.—Severe cases of chilblain are apt to be mistaken for this disease. Chilblains differ from Raynaud's disease, however, in lacking the paroxysmal character, in not all being at the extreme ends of the digits, and in generally showing some signs of inflammatory exudation.

Treatment.—The most important part of the treatment is the prophylaxis, which consists in attending to the digestion and



FIG. 186.—Gangrene of Toes from Raynaud's Disease.
(Boy of 7 years.)

nutrition, and in guarding against any unnecessary exposure. The prolonged use of cod-liver oil and arsenic seems beneficial in chronic cases.

When the attacks occur, the most successful means of relieving the symptoms, especially in acute cases, consist in the use of the galvanic current. The best mode of applying it is described as follows by Sir Thomas Barlow: "Immerse the extremity of the limb which is the subject of local asphyxia in a large basin containing salt and tepid water; one pole of a constant current battery is placed in contact with the upper part of the limb, above the level of the water, and the other pole in the basin, thus converting the salt and water into an electrode. As many elements as the patient can comfortably bear should be

employed; and the current should be made and broken at frequent intervals, so as to get repeated moderate contractions of the limb. The patient should also be instructed to make voluntary movements of the digits while the galvanism is applied."¹ When the two limbs are equally affected, in a typical paroxysmal case, and the electric treatment is applied only to one of them, the limb thus treated is found to recover more rapidly than the other which is merely kept warm. Massage and Swedish movements should also be used. If the pain is severe, or if gangrene threatens, opium is indicated.

Gangrene of the Lower Limbs in Young Infants.

Occasionally gangrene of one or both legs is met with in young babies, following thrombosis of the popliteal artery. In the few such cases we have seen, no cause of the thrombosis was found. All the children died.

¹ Barlow, art. on "Raynaud's Disease," *Allbutt and Rolleston's System of Medicine*, 1910, vii., 120.

CHAPTER XXV

EXAMINATION OF THE LUNGS AND RESPIRATORY PASSAGES

Inspection

Form of the Chest.—The infant's chest differs considerably in shape from the adult's in being more cylindrical, and its section is consequently more nearly circular in outline. Its form is also readily altered by any disease which either lessens the resiliency of the chest-wall, or interferes in any way with the free play of the lungs. The commonest chest deformity met with in young children is that associated with rickets; but we also often find other types due to infantile scurvy, asthma, and adenoids, various degrees of pigeon-breast, and occasionally unilateral retraction or bulging due to disease of the lungs or pleura, or to curvature of the spine.

The **rickety thorax** is characterised by beading of the ribs and by its peculiar shape (Figs. 80 to 83). The *beading* or *rosary* is situated at the junction of the cartilaginous and osseous portions of the ribs, and in fat children is more easily felt than seen. The swellings usually project on the pleural side of the chest-wall a good deal more than on the surface. The first and second ribs are those least affected, while the largest beads are found on the fifth and sixth—that is, on those with the widest range of movement.

In some severe cases of rickets we find a few *posterior beads* on one or other side of the back of the chest (Fig. 86, p. 254). These swellings are asymmetrical in position, and their pathology is quite different from that of the ordinary rickety rosary. They are composed of callus which has formed round fractures of the ribs, caused usually by lateral compression of the chest in lifting the child. Similar lesions of the clavicles are not uncommon and are due to sudden lifting of the patient by his upper arms.

As the chest-wall, outside the line of the beading, is abnormally soft and yielding, it becomes indrawn in this position, causing parallel grooves in front of and behind the rosary. In some rickety chests there is also another horizontal groove running across the front at the level of the upper end of the xiphi-sternum (*Harrison's sulcus*). This is due to imperfect expansion of the bases of the lungs. It is often very well marked in cases of congenital atelectasis. In extreme degrees of rickety deformity the chest assumes a peculiar shape (Figs. 80 to 83), the cyrtometer tracing of which somewhat resembles the outline of a violin (Fig. 187). The thoracic deformity which occurs in some cases of **infantile scurvy** has been already described (p. 232).

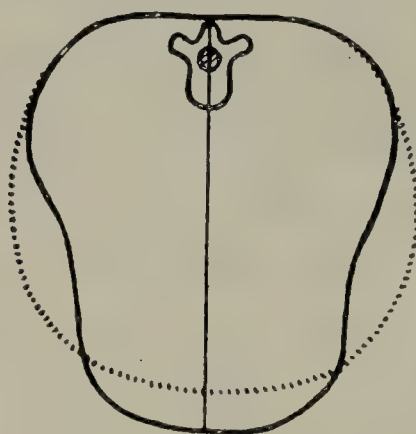


FIG. 187.—Rickety Chest. Dotted line indicates shape of normal chest at same age (Gee).

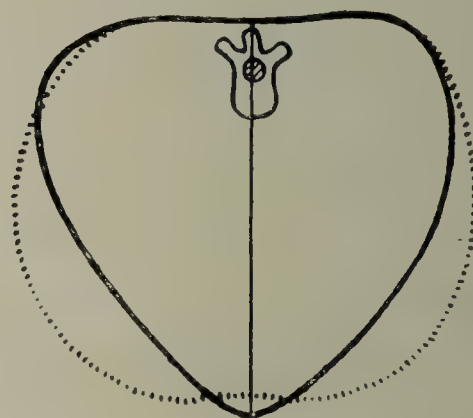


FIG. 188.—Pigeon-Breast. (Child of 7 years.) Dotted line indicates natural shape at same age (Gee).

In scurvy also we sometimes find swellings on one or more ribs due to localised subperiosteal hæmorrhages.

In **pigeon-breast** the abnormal forward projection is more pointed than that in rickets, so that its horizontal outline is sometimes almost triangular (Fig. 188). This deformity may be said to present in a permanent form the shape which the normal child's chest assumes, temporarily, during forcible inspiration through a narrowed glottis (as in croup or whooping-cough). It develops gradually as the result of frequently repeated and long-continued temporary changes of form. In infancy it is seen in congenital atelectasis, in bronchitis with collapse, and to a less extent in congenital laryngeal stridor. One may watch its development, in a previously well-formed chest, during a severe attack of whooping-cough. When pigeon-breast is present in older children there is generally a history of recurrent bronchitis.

The Adenoid Chest.—Practically all cases of adenoids show some chest deformity—the degree varying with the extent to which the growths have interfered with the free entrance of air into the bases of the lungs and restricted the movement of the diaphragm. In severe cases in young children with soft chest-walls, there may be regular pigeon-breast. In older children, however, the deformity is less extreme. It consists in a transverse constriction of the chest about the level of the lower end of the sternum, with a more or less deep vertical mesial groove over the ensiform cartilage and above it.

In some weakly children we meet with a form of chest deformity which is in striking contrast to that of pigeon-breast. In this the thorax is broadened from side to side and much flattened antero-posteriorly. The lower end of the sternum is depressed, so far as to lie at the bottom of a deep hollow, and its posterior surface is approximated to the spinal column. The cyrtometer tracing has a somewhat kidney-shaped outline.

The characteristic *barrel-shaped chest* of asthma often begins to develop in young children; and asymmetrical deformities of the thorax are frequently found as the result of old pleurisy or atelectasis. Many children show a combination of different kinds of thoracic deformity.

All varieties of malformed chests in children have a strong tendency to improve as the child gains strength and the body grows and develops—provided their original causes have ceased to act. This disposition towards recovery can be increased greatly by regular breathing exercises, musical drill, and various forms of gymnastic movements of the upper limbs and trunk. Swimming is probably the best exercise for developing the chest.

In inspecting the chest, marked enlargement of the superficial veins is worthy of note. This is found as a sign of enlarged bronchial glands, and especially in cases of intrathoracic tumour.

Movements of the Chest.—In infancy and early childhood the chest moves little in ordinary breathing compared with the abdomen, and this abdominal type of respiration usually continues till about the seventh year. A notably thoracic type of breathing in young children is characteristic of acidosis. The same thing may also be observed occasionally, to a less extent, for some time after a severe and prolonged attack

of whooping-cough. In watching the respiratory movements we must notice whether the movement is equal on the two sides, and if there is much indrawing of the episternal notch or of the epigastrium on inspiration. Such indrawing means that sufficient air is not entering the chest. Its significance varies according to the circumstances in which it is met. Thus, in extreme rickets, its occurrence may be largely explained by the abnormally collapsible character of the chest-wall, and indicates no immediate risk. In a fairly normal chest, however, the same amount of indrawing may be a serious sign. In broncho-pneumonia the degree of the retraction helps us to gauge the extent to which the lungs are affected; while in croup its presence in a severe degree is an indication for immediate surgical assistance.

In watching the respiration it is well to note if the normal bulging of the upper part of the abdomen occurs with each breath. If it is replaced by a sinking in of the region with each inspiration, it signifies deficient action of the diaphragm.

The movements of the *alæ nasi* should also be observed. In dyspnœa from serious organic disease they are often greatly exaggerated; and if, in a case of noisy or rapid breathing, there is no increased movement of the *alæ*, it is a reassuring sign. We must, of course, remember, in judging of the movements, that the development of the nostrils and their muscles varies much in different children.

Palpation.

One of the first things to be done in examining the *lungs* is to determine the position of the *heart's apex-beat*, and whether there is any distinct epigastric pulsation. If the apex is displaced to one or other side, as often happens in pleurisy with effusion, in collapse, and in fibroid conditions of the lung, this throws a useful light on the diagnosis and may shorten the subsequent examination. In displacement of the heart to the right (*dextro-cardia*) the possibility of transposition of the viscera or a diaphragmatic hernia must be remembered. In cases of acute lung disease, the state of the heart as to strength, or weakness, or dilatation of its right side, is generally far more important from the point of view of the prognosis and treatment than is the extent of lung involved.

Vocal fremitus is sometimes difficult to obtain in children, owing to the quality of the child's voice and his unwillingness to speak out; but it may be easily obtained when he is crying. Rhonchal fremitus is observed in many cases of bronchitis.

Auscultation.

When little children are frightened, they often hold their breath when we try to auscultate them. This may delay our examination, but, at any rate, it shows that there is no serious lung disease present. Screaming is not nearly so troublesome an interference with auscultation as one might expect, for it makes the child take long deep breaths, and thus accentuates any accompaniments there may be. Similarly, the auscultation of the heart may often be carried out quite easily when the child is crying vigorously, because, in the pauses between the cries, there is time for one or two cardiac revolutions to take place. Whimpering and suppressed sobbing interfere far more with our examination.

The child's chest has great powers of conduction, so that the heart-sounds are heard at the back comparatively well without any consolidation of the lungs being present, and crepitations produced on the one side may be heard clearly on the other.

The *breath-sounds* in young babies are naturally very weak, owing to the feeble shallow nature of the breathing. They gain in strength as the child grows, and about the age of six months they have acquired the peculiar harshness characteristic of *puerile breathing*. In older children *abnormal weakness* of the sounds is an important sign. We meet with it in the early stages of pneumonia, in pleuritic effusion, in collapse of the lung, and in pneumothorax. It is also sometimes found in cases with great thoracic deformity, and in these is apparently due to local emphysema or collapse. If the breath-sounds differ in loudness on the two sides of the chest, the side with the weaker breathing is generally the abnormal one. Areas of weak breathing, which pass off rapidly, are sometimes found in children from temporary collapse of a portion of lung due to blocking of a bronchus with mucus.

Tubular breathing is more often met with in pleurisy with effusion in children than in adults, and its presence sometimes leads to a mistaken diagnosis of consolidation. When a large

area of solid lung is present on one side, the tubular breathing to which it gives rise may sometimes be heard so plainly over parts of the normal lung as also to be misleading. In pleurisy in young babies we often fail to hear *friction* sounds, and are better guided to a diagnosis by the catch in the breath and the evident uneasiness which follows when the child inspires freely or attempts to cry.

A peculiar alteration in the rhythm of the breathing is often noticed, and is perplexing to beginners. In ordinary breathing we have inspiration followed by a shorter expiration, then a pause; then inspiration again, and so on. In the peculiar breathing referred to, there is first a long, loud expiration, the noise of which is sometimes accompanied by a sort of grunt. This is followed immediately by a short inspiration, then there is a pause, and then expiration begins again. This *breathing with expiratory rhythm* is heard in its most marked degree in commencing pneumonia. A certain amount of it, however, is common in little children with perfectly healthy lungs—when they are apprehensive or frightened.

Vocal resonance frequently cannot be obtained in the usual way by getting the child to speak, but the cough or cry is generally quite sufficient to elicit it.

Percussion.

In children percussion of the lungs should always be light; for if strong it is apt to mislead by bringing out dullness or resonance, as the case may be, from underlying organs. If the child is being examined in his mother's arms, it is essential *to see that the patient is sitting straight*, because even a slight twist of the spine may give rise to distinct differences in the note on the two sides of the chest, and for this reason it is advisable to attempt the examination of even the youngest child lying. Variations in the curve of the chest-wall have a similar effect; so that, in a deformed chest, local impairment of the percussion note may be found which is not due to any change in the lung, but merely to a sharper curve of the ribs. It is important to remember that in the child the note is less resonant at the left apex in front and at the right base behind than at the corresponding points on the opposite side of the chest.

A slight area of dullness may be due to a local patch of

collapsed lung, or to the result of an old pleurisy. Nevertheless, a slight impairment apart from any other sign or symptom is not to be regarded very seriously in children. Even when fluid is present in the pleura, the dullness is often less absolute than might have been expected; and patches of consolidated lung may cause very little dullness, owing to their being surrounded by emphysema. As a result of the yielding nature of the chest-wall a well-marked *cracked-pot sound* is often obtained on percussion in little children with perfectly healthy lungs—especially when they are crying. When acute pleurisy is present, there may be considerable tenderness on percussion.

Sputum.

Natural expectoration does not usually begin till between five and seven years old. Before this, unless they have been taught otherwise, children always swallow the sputum. If it is important to obtain a specimen from a baby or a young child it can be procured by passing the finger, round which is a piece of gauze, into the pharynx; this sets up coughing and the sputum which is brought up is caught by the swab on the finger before the child has time to swallow it. The sputum collected is immediately spread on a slide and stained in the usual manner.

In this way it is possible to discover the presence of tubercle bacilli in even the youngest infants.¹ In lieu of sputum some writers advise the stomach washings or the fæces. In our experience tubercle bacilli have never been observed in the fæces or stomach washings when they could not be more readily detected in the sputum obtained as described above.

Hæmoptysis is rarely an important or serious symptom in early life, although a little blood-stained expectoration is often seen in cases of pertussis or other violent forms of cough, and also in slighter coughs if the throat or gums are ulcerated. *Hæmoptysis* does not occur in phthisis in children as an early symptom, as it so often does in adults. It is, however, not an uncommon terminal occurrence in pulmonary tuberculosis, in bronchiectasis, and especially in gangrene of the lung.

Fetid sputum is characteristic of pulmonary gangrene, and also of ulcerative stomatitis. In the bronchiectasis of young children the sputum has often, at first, no offensive odour.²

¹ L. Findlay, *Arch. of Pædiatrics*, Feb. 1904, p. 126.

² L. Findlay and S. Graham, *Arch. Dis. Child.*, 1927, ii., 71.

The Larynx.¹

The upper aperture of the larynx at birth (Fig. 189) differs little from that in older children (Fig. 192). Its structures, however, are much softer and more collapsible, so that they are frequently sucked together by the irregular jerky inspirations of the young infant. This soon results in a change of form to the infantile type (Fig. 190), which is found in a varying degree in all young infants who have breathed. In the infantile larynx the epiglottis is distinctly gutter-shaped on its posterior aspect, while the soft and yielding aryepiglottic ligaments come nearer together so as to narrow the upper aperture. The narrowness of the orifice, and the softness of its walls, account

UPPER APERTURE OF THE LARYNX AT DIFFERENT AGES.



FIG. 189.—At birth.



FIG. 190.—At 3 months.

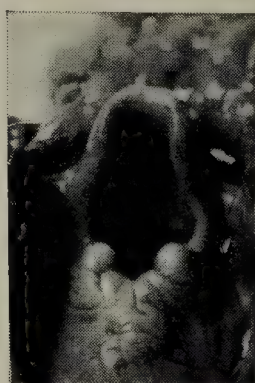


FIG. 191.—At 7 years.

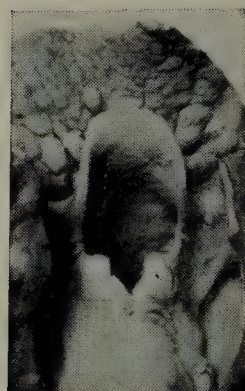


FIG. 192.—At 9 years.

for the readiness with which crowing occurs on slight occasion in young babies.

As the child grows older, the parts become more rigid, and, in most cases, they gradually assume the adult type (Figs. 191 and 192). Occasionally, however, the infantile type persists in some degree even into adult life.

Laryngoscopy.—Even expert laryngologists find it difficult to see the vocal cords in infants of a few months; and it may be quite impossible to do so in the ordinary way, owing to the extreme readiness with which the contents of the œsophagus and stomach are brought up on any attempt to introduce a laryngeal mirror; consequently, a view of the epiglottis is often all that can be obtained. In new-born babies this tendency is not so strong as in those a little older, so that in them the examination is rather easier. Direct laryngoscopy, by Killian's

¹ J. Thomson and Logan Turner, *Brit. Med. Journ.*, 1900, ii., 1561.

or Jackson's tubes, is often successful in skilled hands when the ordinary method fails.

When an ordinary laryngeal examination is necessary in an older child who is very nervous, it may sometimes be rendered easier if a little chloroform is given—just enough to make the child drowsy.

The Breathing.

It is characteristic of the respiration in young infants that it is irregular in rhythm and force, as if the co-ordination of its movements were not yet fully under control; and relatively sudden inspiratory efforts are frequently repeated. When excited, the baby is apt to crow, although he does not do so at other times. A similar, but louder and more persistent crowing, which exactly resembles that in laryngismus stridulus, is frequently heard in young infants while they are coming out of chloroform narcosis; and loud crowing is also a usual accompaniment of operations on the prepuce and anus, owing to the sudden forcible inspiration which is caused by stimulation of the nerves of these parts. Along with all sorts of crowing, there is always indrawing of the lower chest-wall which is usually proportionate to the loudness of the sound.

Rate and Rhythm of the Respiration.—In young infants the rate of the breathing varies so much that it is sometimes difficult to estimate it accurately. It is best counted during sleep, and we should take the average of several minutes. In new-born babies the rate varies from 32 to 50 respirations in the minute, and during the first year from 25 to 35. During the second, third, and fourth years it is about 25 in the minute. The respiration rate, like that of the pulse, varies, not only with the body temperature, but also with the mental state. Very irregular breathing is characteristic of cerebral disease.

Rapid Breathing.—In infants of a month or two old we often find extremely rapid respiration—perhaps 100 to 120 respirations in the minute—without any respiratory distress (*polypnœa*). This may be seen, for example, in pyrexia with acid urine, and its presence does not indicate any respiratory complication. Rapid and deep breathing is also characteristic of food intoxication, especially when acidosis is present.

An increased rate of breathing, if accompanied by dyspnœa, usually indicates respiratory disease, and may be helpful in the

diagnosis of early cases. There are various fallacies, however, to be guarded against. Thus, in cases of cerebral disease, the respiration may appear too rapid because the pulse is abnormally slow. Again, an increased rate of breathing may occur without any lung disease in children with deformed (*e.g.*, rickety) chests, or with great abdominal distension, and also in acute peritonitis. Many infants also, as Henoch pointed out, have a respiration rate of 60 to 90 to the minute without any local cause, during the state of nervous irritability which accompanies teething; in these there is no respiratory distress. Rapid breathing is also seen in acidosis, in some forms of asthma, during and after whooping-cough even when the lungs are not seriously affected, and in measles when the rash is coming out. It is occasionally seen in encephalitis lethargica, and is also a common symptom in early cases of acute cerebro-spinal meningitis and coli-pyelitis.

Pulse-Respiration Ratio.—The pulse-respiration ratio in health should be 1 to $3\frac{1}{2}$ or 4, and any great disturbance of this proportion is of clinical importance. If the proportion is less than 1 to 3 the condition is almost certainly of a pulmonary nature: although pyelonephritis and cerebro-spinal fever, especially during infancy, upset the pulse-respiration ratio this never falls below 1 to 3.

Sound of the Breathing (*Extra-auscultation*).—A great deal can often be made out about the state of a child's respiratory passages by listening to his breathing, his cry, and his cough.

Snuffling breathing indicates obstruction of the nasal passages. This often arises in young infants from ordinary catarrh; but when it goes on for a long time or occurs apart from other catarrhal symptoms, it always suggests the presence of syphilis. "Snuffles" is one of the most constant symptoms of congenital syphilis, and usually appears before the rash (p. 916). Sometimes it is very slight, and it may be inaudible unless the child's mouth is closed. Snuffling is one of the common symptoms in anaphylaxis.

Snoring during sleep, with noisy breathing while awake, and a nasal tone of voice, commonly indicates the presence of swollen tonsils or adenoids, but may also be due to paralysis of the palate. *Noisy breathing* with dyspnoea chiefly during inspiration, and a cry which is nasal but not generally hoarse, is characteristic of retro-pharyngeal abscess, and calls for digital examination of the pharynx.

Deep *sighing* occasionally occurs in little children, without meaning much, but it is an important symptom in the prodromal stage of tuberculous meningitis. Sighing breathing is also characteristic of over-distension of the right side of the heart. Reference may also be made here again to the "grunting" noise which accompanies the dyspnœa of pneumonia and to the somewhat similar noisy panting breathing, with expiratory rhythm, sometimes noticed in cases of masturbation in little children. Noisy gasping respiration is found in certain toxæmic conditions (such as diphtheria and influenza) before death, and indicates the formation of an ante-mortem clot in the heart.

Exaggerated noisy expiration is a very common and characteristic symptom of encephalitis lethargica in children. One of us (J. T.) found it present in the majority of examples of the condition, and a valuable help in diagnosis. Somewhat similar hyperpnœa of a less noisy character occurs in acidosis (p. 559).

Laryngeal or stridulous breathing is due to either organic or spasmodic narrowing of the windpipe, and is met with in true and false croup and in other forms of laryngeal obstruction. In young babies it is frequently a sign of congenital laryngeal stridor, but it may also be caused by laryngeal papilloma, adenoids, and tracheal obstruction from enlarged glands. It is common during the onset of measles.

Bronchial wheezing is often heard in bronchitis of the larger tubes as well as in asthma.

A *hissing* character of the breath-sounds is sometimes noticed in children who are unconscious from uræmia, and is helpful in the diagnosis.

Dyspnœa.

That a child's breathing should be rapid or noisy does not always matter very much; but whenever it ceases to be easy and effortless, and becomes consciously embarrassed, we may be sure that there is something seriously wrong. Different forms of dyspnœa have different characters, according to the various conditions under which they occur. Thus, in some, the shortness of breath is spontaneous, while, in others, it only follows exertion; the difficulty may be inspiratory as in early croup, or expiratory as in asthma; it may be accompanied by acceleration of the respiration as in pneumonia; or by its slowing as in

laryngeal obstruction; in many instances cyanosis is present; in others (*e.g.* acidosis), although the dyspnœa is severe, cyanosis is absent. Cases of abnormal dyspnœa may also be divided into those in which the symptoms are intermittent and those in which they are continuous.

Causation. — *Intermittent dyspnœa* is met with in such conditions as (1) stridulous laryngitis and laryngismus; (2) in asthma; (3) in cases of varying pressure on the trachea and bronchi by enlarged bronchial glands; (4) when there is a moving foreign body in the larynx; (5) finally, painful, rapid, and difficult breathing, setting in acutely at intervals in a young baby, along with deficient entry of air into the bases of the lungs and no other physical signs, is very characteristic of commencing whooping-cough. Its real nature is apt to be overlooked because at this age it is often unaccompanied by anything resembling a whoop.

Continuous dyspnœa is met with under very various conditions.

(1) It may be caused by any *recent narrowing* of the lumen of the upper air-passages, such as acute swelling of the mucous membrane of the nose or nasopharynx, croup, or a fixed foreign body in the windpipe—an intubation-tube may cause it. It may also arise from the pressure of a post-pharyngeal abscess or enlarged bronchial glands.

(2) *Obstruction of the pulmonary circulation* is another cause of embarrassed breathing. It occurs in severe valvular heart disease, and in pericardial effusion, and is a striking symptom in thrombosis of the pulmonary vessels.

(3) Another cause of embarrassed breathing is *pressure on the lungs from without*. Extreme flatulent distension of the abdomen may give rise to it; and it may follow pneumothorax and large pleural effusions; though, if the latter form slowly, the dyspnœa may only occur on exertion.

(4) *Acute collapse of a lung*, such as may occur in progressive muscular atrophy, produces severe dyspnœa; and a lesser degree results from the partial collapse of the bases which may follow paralysis of the diaphragm.

(5) *Acute respiratory disease* is a very common cause of difficult breathing. We see it in severe bronchitis and broncho-pneumonia, in emphysema, and in croupous pneumonia. It is less marked in phthisis, because the limitation of the air space takes place so gradually.

(6) In *Bright's disease with heart-failure*, dyspnœa is a frequent and urgent symptom.

(7) An interesting and important form of distressed breathing is the *hyperpnœa* or "air-hunger" seen in acidosis. In this the respiration is deep and laboured and may be increased in rate, but there is no cyanosis. The breathing is also peculiar in having a strikingly thoracic type, very unlike that usually seen in childhood. This type of breathing is sometimes very useful in drawing attention to the unexpected presence of diabetes, salicylate poisoning, or some other variety of acidosis. Its significance is dealt with elsewhere (Chapter XXIII, p. 551).

(8) In all forms of *anæmia* dyspnœa on exertion is apt to occur.

The Cry.

Much may be learned from listening to a child's cry. From its loudness we can gauge his strength to a certain extent, and if he cries vigorously and long, without getting out of breath, we may be almost sure that he has no serious acute disease of his lungs or bronchi. If a child with a large area of dullness cries loudly, this is in favour of his having a pleural effusion rather than pulmonary disease. Occasionally, however, children with acute pneumonia can make a good deal of noise.

The Cough.

Coughs vary greatly in character and significance. While the presence of a cough naturally draws attention to the chest, it may prove to be due to some morbid condition elsewhere (in ear, brain, heart, or stomach); and, when from the respiratory tract, it is quite as often a sign of irritation in the larynx or trachea, or about the pharynx and its neighbourhood (tonsils, uvula, or adenoids) as of disease lower down.

The character of the cough is sometimes useful in indicating its source. It is loud and clanging at the beginning of an attack of croup, and husky and stridulous at a later stage. In bronchitis it is often deep and harsh. In pneumonia with accompanying pleurisy it is suppressed and painful. If a child coughs loudly without wincing, we may be sure that he has no acute pleurisy. A very annoying and persistent cough sometimes occurs when a pleural effusion is beginning

in a case of chronic lung disease. Measles, in its early stage, is a common cause of constant uncontrollable coughing. When a child suffers from a loud noisy cough on lying down at night and also when he wakes in the morning, although he is not much troubled with it during the day, he probably has a catarrhal condition of his throat, with or without dyspepsia.

Often the cough has a distinctly paroxysmal character. This is most marked in whooping-cough, but is also frequently present in some degree in influenza and in severe bronchitis. A similar cough occurs in some cases of empyema, of enlarged bronchial glands, and of adenoids. A cough, worst at night, occurring in paroxysms and ending in vomiting, nearly always indicates whooping-cough; and if there is puffiness of the eyelids, or an ulcer under the tongue, the diagnosis is strongly confirmed. When a severe suffocative cough occurs in a young baby, it is always well to think of whooping-cough as possible, even although there is no distinct whoop present.

Treatment of Cough.—The proper treatment for a cough is, of course, that of the morbid condition which is causing it; but, where the cause cannot be ascertained, we must treat the cough as a symptom. Sometimes considerable relief is afforded by a large linseed and mustard poultice applied round the chest, or a small mustard plaster over the trachea or at the root of the neck. A hot drink and a steam-kettle are also to be recommended. In cases where, as in whooping-cough, the cough is violent, the support of an abdominal belt often gives the child great relief. We may also use such internal remedies as codein, heroin, antipyrine, aspirin, or butyl-chloral.

Sneezing.

Sneezing is, of course, a common symptom in ordinary catarrhs, in some forms of influenza, and especially during the invasion of measles. Severe and persistent sneezing during the summer months in children over five years may indicate hay-fever; and fits of sneezing are often noticed in children with ordinary asthma, and in those who have idiosyncrasies for certain foods.

Hiccough.

As hiccough is due to spasmodic action of the respiratory muscles it may be dealt with here, although it is chiefly connected with the digestive organs. In children, as in adults,

it occurs sometimes in the last stage of serious diseases of the alimentary system, such as appendicitis and peritonitis; and also in some nervous conditions. Generally, however, we meet with it as a normal phenomenon in healthy infants. According to Thévenet,¹ it is especially frequent in breast-fed babies during the first three months of life, becomes less common as they grow older, and is rather rare by the end of the first year. It is less often seen in bottle-fed infants and occurs later in them. It usually comes on after a feed, and, when it does so, it may be regarded as the sign of a satisfied stomach comfortably digesting a copious meal; the administration of more food checks it. If the digestion is disturbed the hiccough ceases, and only returns when the derangement passes off. It does not occur when the stomach is empty. Hiccough, in babies, often follows regurgitation and sometimes comes after vomiting; but its recurrence is generally a reassuring sign and indicates that the last meal taken is going to be retained.

¹ *Lyon Méd.*, 27th Aug. 1905, cv., 333.

CHAPTER XXVI

DISEASES OF THE RESPIRATORY SYSTEM

Pneumonia

PNEUMONIA may be either a primary disease or occur as a complication of, or be secondary to, some other infection. The latter variety is invariably of a broncho-pneumonic nature, but when pneumonia occurs as a primary disease, it may be either croupous (lobar) or catarrhal (broncho-pneumonic) in type.

Most writers hold that as a primary disease both these types of pneumonia may occur at any age during childhood, although it is generally admitted that while the croupous variety is usually met with during later childhood, the catarrhal type shows a predilection for infancy and the very early years of life. McNeil and his co-workers,¹ however, suggest that, as a primary condition of the lung, lobar pneumonia is more common than broncho-pneumonia, even during the early years of life. This opinion they base on a very extensive recent investigation of both clinical and pathological material. While admitting that a clinical differentiation between the two types in the early years of life is often impossible, they estimated that of 253 examples of primary pneumonia passing through one ward of the Children's Hospital, Edinburgh, between the years 1921 and 1928, 143 (*i.e.* 56 per cent.) were of the lobar type.

Croupous or Lobar Pneumonia.

The question of the age at which lobar pneumonia first occurs cannot, in our opinion, be decided on clinical grounds. Clinically, the picture may be typical of a lobar pneumonia (a sudden onset of the illness, consolidation limited to one lobe or the greater part of one lobe, and a termination by crisis) and yet, when the case comes to post-mortem examination, histologically the lung lesion is found to be catarrhal in type. The

¹ C. McNeil, A. R. MacGregor, and W. A. Alexander, *Arch. Dis. Child.*, 1929, iv., 12.

broncho-pneumonic patches have more or less coalesced and involve almost a whole lobe, suggesting to the naked-eye hepatisation, but microscopic examination will show the lesion to be lobular in distribution, the cellular foci being separated by areas of emphysema with severe involvement of the bronchioles.

The argument that lobar pneumonia is seldom fatal in infancy, and hence post-mortem evidence of no value in deciding the issue, is hardly worth consideration. Lobar pneumonia after three years of age is often subject to fatal complications (empyema, meningitis, etc.) and it is incomprehensible that such should not also occur during the earlier years of life and give opportunity of observing the lesion in the post-mortem room.

Dr Olive Somerville, working at the R.H.S.C., Glasgow, analysed the post-mortem examples of pneumonia occurring between the years 1915 and 1926 and found that they had been classified by the Pathologist as follows:—

Age Incidence of Various Types of Pneumonia (Post-mortem Material, R.H.S.C., Glasgow).

Type.	Age-Period.	
	0 to 3 Years.	3 to 12 Years.
Primary broncho-pneumonia	210	7
Secondary „	232	19
Influenzal „	28	8
Lobar pneumonia	2	14

This analysis shows the rarity with which lobar pneumonia is met with in the post-mortem room under three years of age. Since, however, in most cases the diagnosis of the particular type of the disease had been made from the naked-eye appearances alone, and this was so in the two examples of reputed lobar pneumonia occurring during the age-period 0 to 3 years, Dr Somerville¹ investigated, from the point of view of the minute anatomy of the lesion, a series of thirty-six examples of primary pneumonia. In only one case, a boy of five years, did she find the lesion typical in all respects of lobar pneumonia. In two children under three years of age a mixed type of lesion, partly catarrhal and partly croupous, was present, a condition which we have since found also occurs in children slightly over three years.

¹ O. Somerville, *Arch. Dis. Child.*, 1928, iii., 194.

It would thus seem that during infancy a catarrhal pneumonia is the rule, after three or four years of age croupous pneumonia is the prevalent lesion, and that, between these ages, there is a period of transition during which both types of reaction occur. M. Valleix¹ as long ago as 1850 drew attention to this same point; only he put the transitional period between two and six years.

The mortality from primary pneumonia also supports the idea of some change taking place about this same age-period (Table below). Although the death-rate steadily declines during the first two or three years, there occurs a sudden and marked fall in the percentage mortality during the fifth year of life, which is just subsequent to the age-period when post-mortem evidence reveals a complete reversal in the proportion of the broncho-pneumonic and lobar forms of the disease.

Mortality in Primary Pneumonia (R.H.S.C., Glasgow., 1915 to 1929).

Age.	No.	Percentage Mortality.
0 to 6 months	318	65.4
6 months to 1 year	499	44.8
1 to 2 years	727	28.3
2 „ 3 „	352	15.9
3 „ 4 „	150	16.0
4 „ 5 „	108	3.7
5 „ 6 „	85	8.2
6 „ 7 „	71	9.8
7 „ 8 „	73	6.5
8 „ 9 „	50	6.0
9 „ 10 „	48	6.2
10 „ 14 „	63	3.1

The cause of this different reaction in the lung is not one of the specific agent, as both primary broncho-pneumonia and lobar pneumonia may be caused by the pneumococcus.² It is

¹ M. Valleix, *Amer. Journ. Med. Sci.*, 1850, xix., 211.

² In this connection the bacteriological investigations (unpublished) of Dr Blacklock at R.H.S.C., Glasgow, in pneumonic lesions regarding the type of pneumococcus present are of interest. Dr Blacklock found that pneumococci of fixed types (I, II and III) were seldom isolated, the great majority being of the undifferentiated type (X). From the pleural exudate in post-pneumonic empyema, pneumococci of types I and II were obtained in only 17.2 per cent. of the cases under two years of age, but in 66.7 per cent. in the patients between two and five years, and in 92 per cent. of those between five and thirteen years of age.

much more likely to be due to something inherent in the child, perhaps of the nature of a metabolic or serological peculiarity. As Dr Somerville says, "We are quite familiar with the varying susceptibility of different organs to a specific organism, *e.g.*, the meningococcus; with the varying susceptibility at different age-periods of any individual organ to a specific organism, *e.g.*, the rheumatic infection; with the varying type of reaction at different ages to a specific organism, *e.g.*, the tubercle bacillus; and with the varying susceptibility of an organ to an infection which occurs under special or unusual circumstances. Of the last phenomenon the immunity of the cerebrum to the *Treponema pallida* in a person subject to malaria may be cited."

As a result of the study of their pathological material, McNeil and his co-workers¹ have reached a different conclusion to the above. These observers studied their material in sections of whole lung as well as by the usual histological technique. They took as their criteria for diagnosis limitation of the inflammation to the alveoli to signify lobar or alveolar pneumonia, and involvement of the bronchi and interstitial tissue to indicate the broncho-pneumonic or interstitial type of the mischief. They also remark that difficulties in differentiation arose from the fact that cases occurred which presented the characters of both types of lesion. This they found especially frequent during the first two years of life. However, of 163 post-mortem examples of pneumonia which they investigated, they concluded that 140 were broncho-pneumonic, and 23, of which 18 occurred under two years of age, were lobar in type.

Symptoms.—The child takes ill suddenly, and the earliest manifestations of the disease are usually not respiratory symptoms; they may be vomiting or diarrhoea; headache is also common and in some cases convulsions occur, but rigors are rarely met with, and only in older children. From the beginning the child is generally prostrated. Cough is not usually a very prominent symptom; and, when present, the child tries to suppress it because of the pain it causes. The sputum rarely gives any assistance, and is difficult to obtain. Herpes round the mouth and a vivid flush on one or both cheeks are commonly seen. Sometimes there is distinctly

¹ C. McNeil, A. R. MacGregor, and W. A. Alexander, *Arch. Dis. Child.*, 1929, iv., 85.

localised pain on one or other side, and when present this is very helpful in localising the site of the pneumonia. Pain from the diaphragmatic surface of the lung and pleura may be referred to the shoulder or above the clavicle in front; and, when the disease is elsewhere in the lower lobes, to the abdominal wall. Pneumonia of the right lower lobe often gives rise to pain not unlike that from inflammation of a high appendix. Occasionally, pain in the right side of the abdomen is due to a slight degree of pneumococcal peritonitis.

The breathing is characteristic in various ways. Its rate may not be increased when the temperature first rises; but it is usually markedly so before any physical signs develop. The respirations are generally 50 to 60 or more to the minute, even if only a small portion of the lung is affected; they are shallow and sometimes comparatively easy. The breathing is chiefly abdominal, while in broncho-pneumonia and bronchitis it is more thoracic. Another characteristic peculiarity of the breathing is its expiratory rhythm, which is often accompanied by noisy grunting expiration. The pulse-respiration ratio is always altered.

The temperature curve in children is much as in adults, showing a sudden rise, a continued type, and a complete crisis. The crisis generally occurs between the fifth and ninth days; but it may set in as early as the second or third, and is often deferred to the tenth or twelfth day or even later—especially when the disease spreads and fresh areas of the lung are affected (Fig. 193). In some children marked remissions are present during the whole course of the disease, but these are specially common towards the end of the fever; but all varieties of ending may occur, from the usual typical crisis to a regular lysis. Should the temperature keep up, or if it rises again soon after it has fallen, empyema may be suspected, if no otitis media is present.

The frequency and severity of cerebral symptoms in pneumonia in children is a fact of practical importance. One of the most striking features of lobar pneumonia during childhood is the acuteness of the illness and toxicity in the early days of the infection, and the comparative absence of these during the later days. Thus at the onset there may be wild delirium, while during the last days of the illness the patient appears so comfortable that, were it not for the evidence of the

fever, one would have a difficulty in believing that he were really ill. Another peculiarity of the disease in childhood is the tendency for delirium to appear after the crisis.

When the cerebral symptoms are severe, and the chest signs are delayed, there is a danger of the case being mistaken

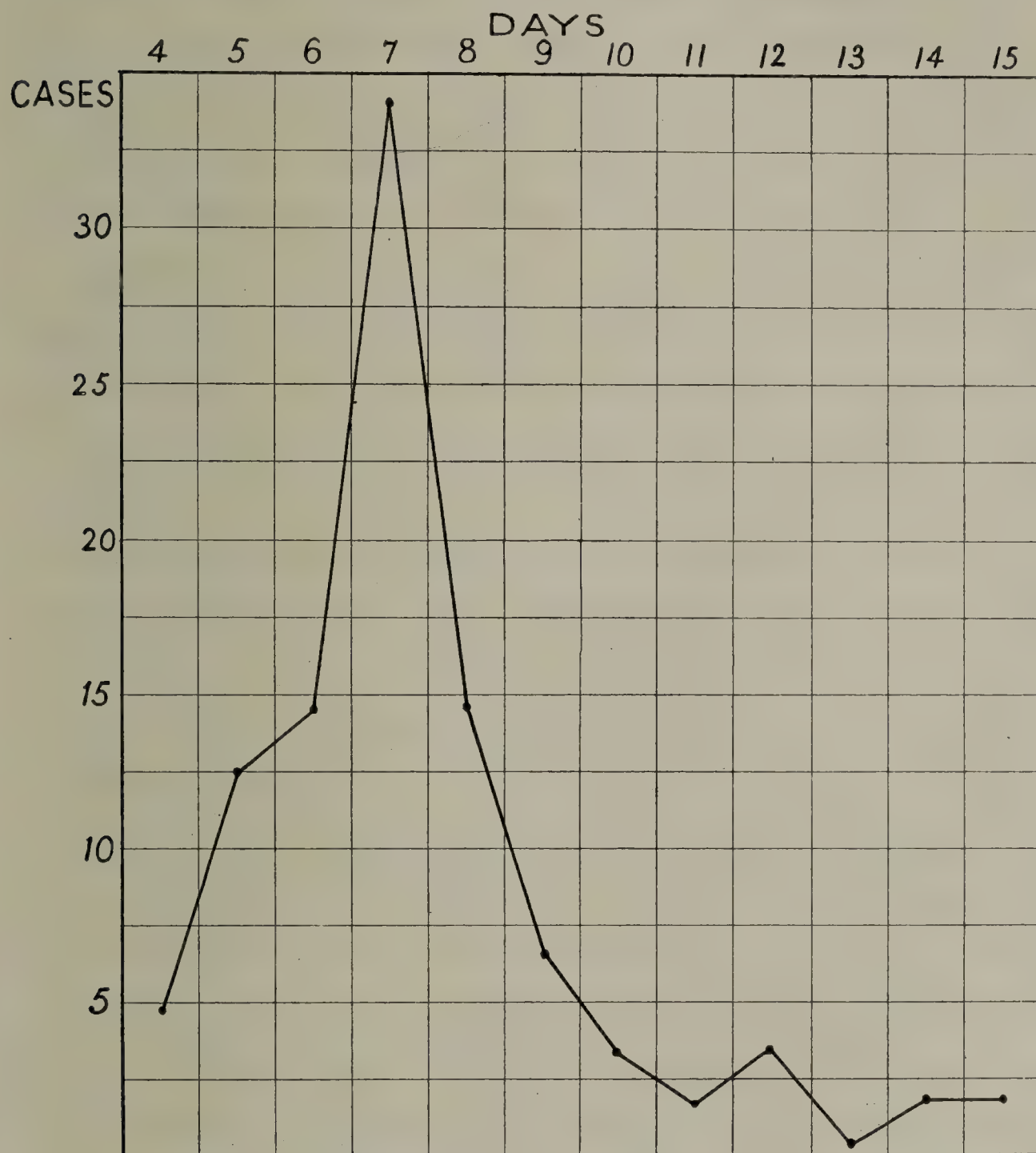


FIG. 193.—Showing day on which crisis occurred in one hundred cases of Lobar Pneumonia in childhood.

for one of meningitis. It may, indeed, be very difficult to be sure, without a lumbar puncture, whether the cerebral symptoms accompanying an undoubted pneumonia do, or do not, indicate the presence of meningitis also. When meningitis is present the cerebro-spinal fluid is always altered in character and increased in amount, whereas in uncomplicated pneumonia the

cerebro-spinal fluid is perhaps under increased pressure but is otherwise normal. This meningeal state is called *meningism*.

The physical signs of pneumonic consolidation are the same in children as in adults (Fig. 195, p. 625). They are generally distinct by the fourth day; but, in some cases, careful and repeated examination fails to discover any before the sixth, or even the seventh day. In such cases the acceleration of the breathing is usually also long of beginning. In children with high fever, digestive disturbances, and irritation about the gums, one should continue, as long as the fever lasts, to suspect the possible presence of a patch of pneumonia and examine repeatedly for it.

Complications. — The most important complication of pneumonia is empyema (p. 635). Otitis media is probably the commonest, and it often occasions no pain. Pneumococcal pericarditis is rare in older children; but in young infants it is a not uncommon cause of death, though it is very often overlooked owing to the absence of friction. Pneumococcal vegetative endocarditis sometimes develops. Pneumococcal meningitis and peritonitis, as already mentioned, occasionally occur. Gastro-enteritis, nephritis, and arthritis are also sometimes met with; and, in cachectic children, gangrene of the lung may supervene. Rarely, temporary paralysis of the sixth cranial nerve occurs. Occasionally abscesses form in the retro-peritoneal connective tissue or elsewhere, as the result of pneumococcal infection; and, whenever obscure signs of inflammation in any situation are found within a few weeks of an attack of pneumonia, the probability of their being due to pneumococcus infection should be borne in mind.

The **prognosis** in croupous pneumonia in older children, who have been previously healthy, is very favourable, considering how ill the child generally seems to be. When once a diagnosis of croupous pneumonia is made there is, as a rule, no more cause for anxiety. Unless he is very feeble, or the disease complicated, or a large area of one or both lungs affected, the case is almost certain to end in recovery. When death occurs in simple pneumonia it is nearly always due to heart-failure. The great importance of the characters of the pulse in the prognosis of this disease has already been discussed (p. 574).

In some children pneumonic consolidation persists for many weeks or even for months, and, after arousing suspicions of

tuberculosis, clears up entirely. Children are not uncommonly met with who, though otherwise robust, have a special liability to pneumonia and who, during the course of a few years, have had three or four attacks.

In children in whom croupous pneumonia occurs as a complication of measles, and in some epidemics of influenza, also in young children who have a lowered power of resistance, the prognosis must be very guarded. Under these circumstances the disease is apt to spread to the other lung, to be followed by empyema, pericarditis, pneumococcal meningitis, or some other serious complication.

Treatment.—Most sporadic cases of croupous pneumonia, in previously healthy children, are not serious and need no special treatment. The disease is self-limited and has a strong tendency to recover; and all that is usually necessary is that the child should be kept lying in bed in a well-ventilated room, or in mild weather in the open air, and be regularly and judiciously fed and nursed. Over-feeding must be avoided, alcohol is unnecessary, and sitting up should be discouraged except in slight cases. Complications and special symptoms have to be watched for and treated when they arise.

The main symptoms which require active treatment are specially high temperature, pain, sleeplessness, cough, and threatening heart-failure.¹

(a) *Pyrexia*.—Generally the fever requires no special treatment; but if the temperature remains for long over 104.5° , or if hyperpyrexia occurs at any stage, something must be done to reduce it. Otherwise there is a risk in the former case of the heart-muscle suffering, and in the latter of convulsions setting in. A specially high temperature sometimes occurs at the beginning of the attack, and this is usually best treated by a dose of Dover's powder. In the later stages of the disease opium is to be avoided, and the administration of alcohol in moderation may be indicated, along with tepid sponging or a cold pack. It is a good practice to use cold sponging every four hours or so if the temperature registers in the axilla more than 102.5° F. This measure will make the child more comfortable and conduce towards sleep. Coutts recommends a rectal injection of cold water (75° F.). Antipyretic drugs should rarely

¹ J. A. Coutts, "Some Observations on the Occurrence and Treatment of Lobar Pneumonia in Young Children," *Edin. Med. Journ.*, Sept. 1902, xii., 209.

be used. Prolonged pyrexia and hyperpyrexia may both be caused by otitis media.

(*b*) *Pain*.—This is generally best treated by a linseed poultice with or without mustard or by hot fomentations. Severe pain at the beginning in older children generally yields rapidly to a dose of Dover's powder.

(*c*) *Insomnia*.—In older children in the early days of the disease, Dover's powder is also useful in insomnia. Later, if the patient is collapsed, alcohol is indicated. If the pulse is fairly good, antipyrine may be given cautiously, and is sometimes very successful. The sleeplessness, especially in young children, may be due to a loaded bowel, in which case a warm enema or a dose of castor oil, forms the best treatment.

(*d*) *Cough*.—The cough may be treated by poultices or a steam-kettle. Syrup of Codeia or Extract of Thyme may be freely given, and often have a marked effect on the cough. Should bronchitis be present, as happens much more frequently in children than in adults, an ordinary ipecacuanha and ammonia cough mixture, with or without paregoric, should be given (Appendix E, Form. 14 to 17).

(*e*) *Heart-failure*.—When this threatens, it should be energetically dealt with by alcohol, caffeine, ammonia, or ether. In severe cases the child should be kept constantly lying and not allowed to sit up. When the right side of the heart shows signs of dilatation and the liver is enlarging from passive congestion, venesection and the application of from four to eight leeches over the præcordium or right hypochondrium are of the greatest value, and one or other should always be employed. A calomel purge is also often useful. When there is much cyanosis, the inhalation of oxygen often gives great relief.

(*f*) *Flatulence*.—If there is much flatulent distension of the abdomen it may be necessary to stop all milk for a time.

No single therapeutic measure in the treatment of pneumonia has such a powerful effect for good as the free use of open air, and the patients do best who are nursed near a widely open window or, better still, on a balcony.

We have no experience of the use of anti-pneumococcus serum in the lobar pneumonia of childhood, but from the fact that in many cases the pneumococcus isolated is of the undifferentiated type, little benefit can be expected from its use (see footnote, p. 616).

Broncho-Pneumonia.

Broncho-pneumonia may occur as a primary disease, or it may be secondary to such infections as whooping-cough, measles, influenza, and diphtheria, or to gastro-intestinal disorders, and it is very commonly the cause of death in marantic and debilitated infants.

We have already remarked that it is this form of pneumonia which almost invariably occurs during infancy and early childhood, although the symptomatology may in many instances closely resemble that met with in the lobar form of the disease. In our experience age alone is the differentiating feature, no matter what the symptoms and result may be. It may be stated that under three years of age the pneumonic condition is almost certainly of the broncho-pneumonic form and over that age most probably of the lobar variety.

Symptoms.—The mode of onset varies considerably in different cases. Frequently it sets in as the culmination of a bronchial or upper respiratory infection; often, however, the pneumonic symptoms come on quite suddenly without previous bronchitis, and there are high fever, vomiting, and sometimes a convulsion as in croupous pneumonia. The symptoms, however, are usually much more obviously pulmonary in character in this kind of pneumonia. The respiration is rapid and laboured, and has a distinctly thoracic type; there is also more or less inspiratory indrawing of the lower lateral regions of the chest-wall. Cough is generally present—shallow, short, and hacking in character. The child is usually too ill to sit up or speak, and he gets short of breath upon the least exertion. Cyanosis is noticeable in severe cases.

The type and duration of the fever are exceedingly variable. In some instances it is high and sustained but of short duration, and ends by crisis. In other cases it is intermittent, of long duration (we have seen it continue for sixteen weeks), and disappears by lysis. In broncho-pneumonia one should never attempt to predict when recovery is to be expected. The rise of temperature, however, and especially in the delicate and premature infant, may be very slight or even absent.

The physical signs, like the course of the disease, are also very variable. These depend entirely on whether the lesion is generalised or confined to one part of the lung. There may be

evidence of the limitation of the mischief to one lobe, or to a part of one lobe (Fig. 194), with dullness to percussion, and tubular breathing. It is these cases which are generally considered examples of lobar pneumonia. On the other hand, the physical signs are often most indefinite, and if the disease is widespread it may simulate bronchitis. The definitely moist character of

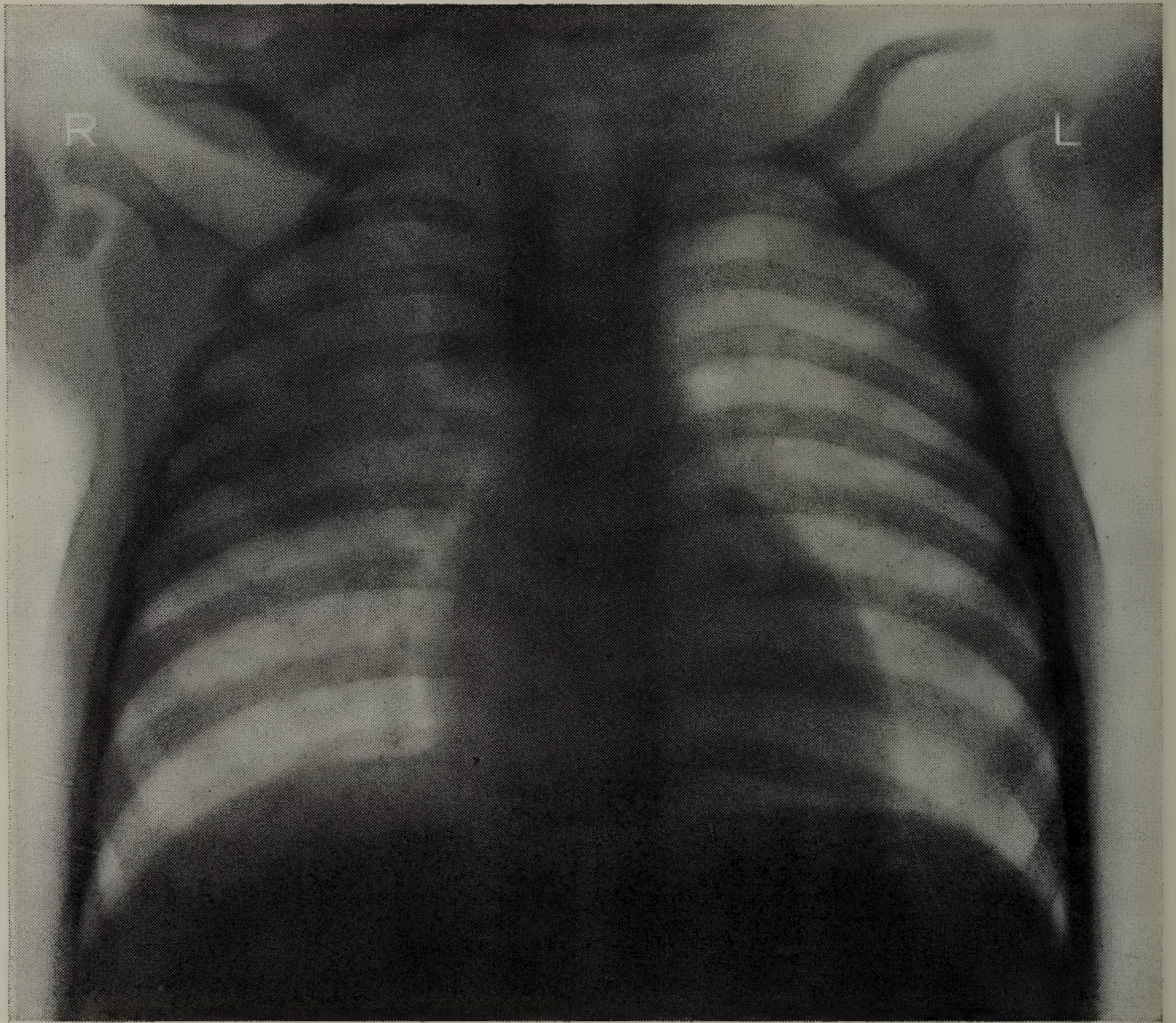


FIG. 194.—Skiagram in case of localised Broncho-pneumonia of right upper lobe in child of 48 weeks.

the râles and the degree of illness of the patient are, however, usually sufficient to suggest broncho-pneumonia even in the absence of any detectable areas of consolidation. This is the condition which was formerly called *capillary bronchitis*. As a rule, however, one or several patches of consolidation make their appearance, as evidenced by dullness, tubular breathing, and a particularly discrete type of râle. The widespread mischief is more characteristic of the secondary varieties, and the localised of the primary varieties of the disease. A word of warning may be given regarding diagnosing as pneumonia questionable areas

of consolidation which are here to-day and away to-morrow, and wander about the lung from place to place. In our experience such cases have generally turned out to be something quite different (infantile paralysis, enteric fever, pyelonephritis, etc.). And finally, it must be borne in mind that pneumonia may be present in the very young and debilitated

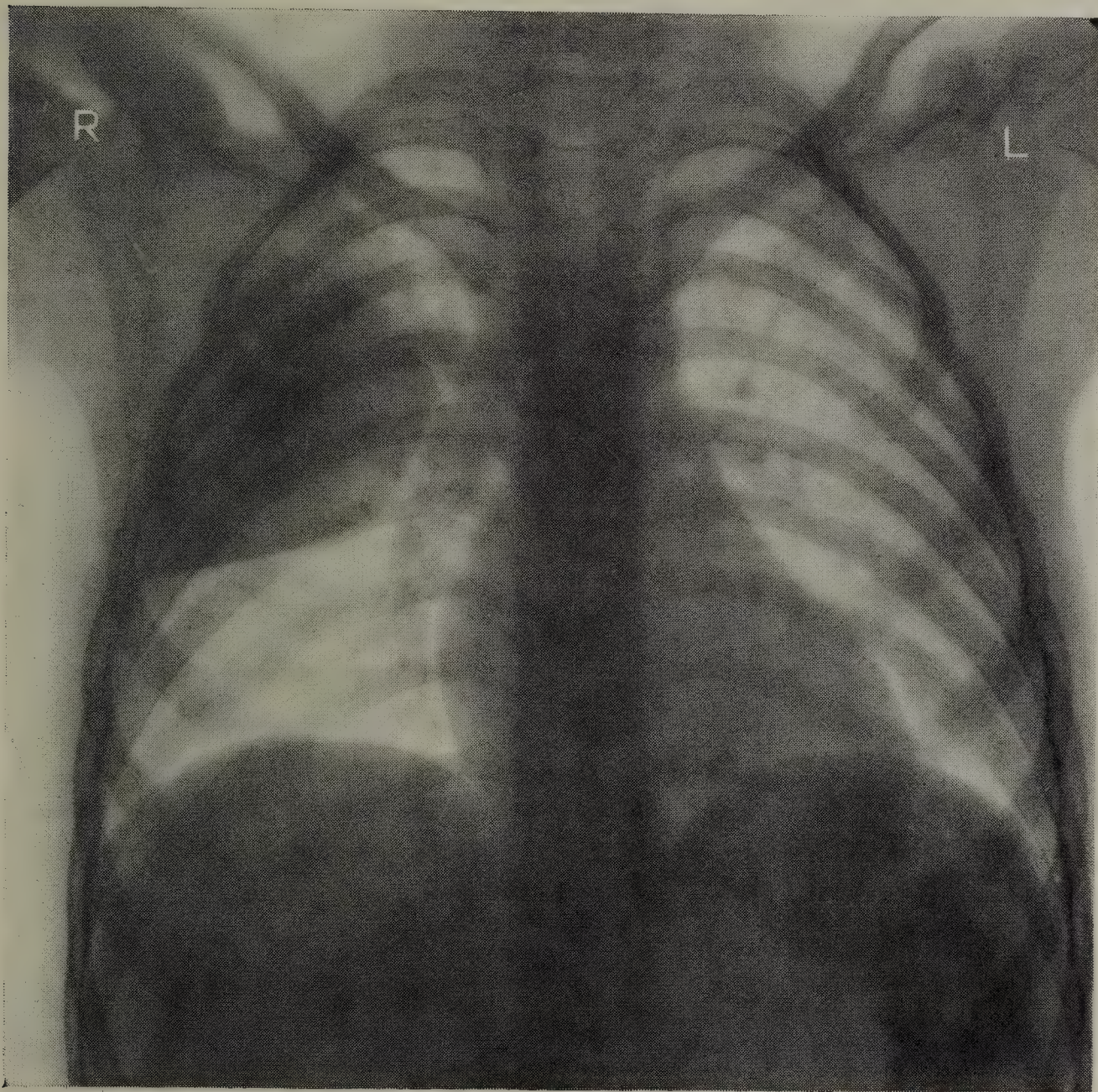


FIG. 195.—Skiagram in case of Lobar Pneumonia (right upper lobe).
(Boy aged 9 years.)

child and give rise to no signs and even be unrecognisable by radiology. The frequency with which this fact is demonstrated in the post-mortem room is one of the most humiliating lessons which the pædiatrician learns.

Complications.—Pleurisy is a less invariable occurrence in broncho-pneumonia than in the croupous form of the disease. It is met with, however, in most cases where the lung is extensively involved, and empyema often follows. Collapse

and emphysema frequently occur, and gangrene of the lung is not very rare in cachectic children.

Diagnosis.—Broncho-pneumonia is to be distinguished from bronchitis by the general symptoms rather than by the physical signs, excepting perhaps the presence of a discrete râle, which is almost invariable evidence of consolidation. The higher range of the temperature, the greater rapidity of the breathing, the shorter and shallower nature of the cough, the indrawing of the chest-wall, the greater prostration, and the marked cyanosis are all indications of the graver disease.

It is not always easy to diagnose broncho-pneumonia from collapse, and, when both are present, to determine what proportion of the symptoms is attributable to each. Atelectasis is characterised by a low temperature, more or less marked cyanosis, and generally indefinite physical signs.

It may be quite impossible to discriminate, at an early stage, between an attack of simple and one of tuberculous broncho-pneumonia. The symptoms and physical signs may be practically identical in the two conditions. As a rule, however, the physical signs are more marked in the non-tuberculous lesion. The occurrence of previous wasting, and especially the history of an attack of measles or whooping-cough some months before the present illness, are suggestive of tuberculosis. The X-ray appearances are of great help in the diagnosis, and of course an examination of the sputum. This question is further discussed at p. 969.

Prognosis.—In forming a prognosis in a case of broncho-pneumonia we must consider the following points:—

(a) The child's age—the younger he is, the more serious is the case (see Table, p. 616);

(b) The state of his nutrition—the chances are *far better in breast-fed* than in bottle-fed babies;

(c) The state of the pulse—if the tension is low, it is a very bad omen;

(d) The form and consistence of the chest-wall—weak muscles and a soft-walled or deformed thorax greatly increase the danger;

(e) The extent of lung involved;

(f) If the pneumonia is secondary, the nature of the primary disease is important—*e.g.*, an attack of broncho-

pneumonia after measles is much more likely to be fatal than a primary attack of apparently equal severity.

Treatment.—The main indications are as follows:—

(a) *Keep the patient warm* in bed and supply him with *plenty of fresh, cool air*. The old-fashioned plan of having the windows shut for fear of a draught is a serious mistake. Draughts can easily be kept off by the use of screens, and so long as the child's extremities are warm, he will (if he has no laryngitis) get nothing but good from abundance of cold, fresh air.

The great value of fresh air in the treatment of this disease was forcibly pointed out in 1904 by Dr Claude B. Ker¹ in an important paper on the results of open-air treatment in the management of broncho-pneumonia complicating whooping-cough. Since then, many testimonies to its efficacy have been given in this country and in America.

When the windows are kept widely open, the appetite and digestion improve, and, with them, the general strength. The nervous tone is also better, and the child is less fretful and sleeps far more, and more soundly.

(b) *See that the nasal passages are as clear as possible*. If there is any nasal catarrh it is most important, especially in young infants, to use a simple alkaline and antiseptic lotion for the nose (Appendix E, Form. 35). Any nasal obstruction is apt to favour pulmonary collapse.

(c) *Avoid embarrassing the respiration* by heavy or tight clothing, and *encourage expectoration*. The continuous application of heavy poultices is not advisable; but an occasional mustard and linseed poultice followed by a light cotton-wool jacket, which must be frequently renewed, is often useful. A simple alkaline expectorant mixture (Appendix E, Form. 14) is usually distinctly helpful in rendering the phlegm more liquid and the cough easier. Moistening of the air, by the use of a steam-kettle or by hanging about the room towels wrung out of hot water, is also sometimes useful—mainly if the upper air-passages are affected. Prolonged use of steam is disadvantageous—it has a devitalising effect. The best results are got when it is used intermittently, one hour on and two hours off, but particular attention should be paid to whether the child is helped by the steam, *e.g.*, sleeps more comfortably.

¹ *Scot. Med. and Surg. Journ.*, Jan. 1904, 33.

In fairly strong children, with slight pneumonia and much bronchitis, an occasional emetic may help considerably, but often the patient is too weak for such treatment. Opiates and most other sedatives are *strongly contra-indicated*. If there is much generalised bronchitis, with abundant râle, the administration of large doses of extract of belladonna ($\frac{1}{4}$ gr. every three or four hours), as recommended by J. A. Coutts,¹ or of atropine ($\frac{1}{100}$ to $\frac{1}{200}$ gr.) hypodermically is often of the greatest benefit.

(d) *Conserve the general strength* in every possible way. Do not upset the digestion by over-feeding; give liquid food in small quantities at regular intervals. If a weakly infant refuses to take enough nourishment, he should be fed by a tube through the mouth or nose.² Keep the baby lying flat, and do not let him tire himself by any unnecessary exertion. It is well, however, to have his position changed from time to time, and, in slight cases, to let him sit up now and then, so as to encourage free inspirations. It is particularly important never to weary the patient by any unnecessary physical examination. For the same reason the clothing should be such that it can be removed with as little exertion as possible on the part of the child. A cotton-wool jacket and an arrangement of shawls is far better than a night-dress with sleeves.

(e) *Ward off collapse* by stimulant measures. In many, especially secondary cases, alcohol is advisable, and small doses of strychnine may sometimes be useful. Inhalation of oxygen is undoubtedly beneficial. Geoffrey Bourne³ has devised a special holder to enable the oxygen to be given continuously by means of a nasal catheter.⁴ When collapse threatens, a mustard pack is often of great service. Should the right side of the heart become dilated, leeching or venesection may be called for. This is, however, very seldom indicated in this form of pneumonia.

(f) *If reduction of the temperature* is required, cold sponging is preferable to the use of drugs.

(g) *Make sure of a complete convalescence* by the use of cod-liver oil and tonics; and especially by sending the child to the country for a time.

¹ J. A. Coutts, *Brit. Med. Journ.*, 1899, i., 207.

² Ibrahim, *Deutsch. Med. Wochenschr.*, 1910, No. 23, xxxvi., 1081.

³ G. Bourne, *Lancet*, 1922, ii., 23; and *Brit. Med. Journ.*, 1922, ii., 40.

⁴ The apparatus is made by Messrs Maw Son & Sons, London.

(h) *Serum therapy.* In view of the fact that the etiological organism in the vast majority of cases is of the undifferentiated type (p. 616), serum therapy is little likely to be of benefit.

Collapse of the Lung.

Collapse of the lung is a common and important incident in the course of all kinds of respiratory disease in infancy. It is also frequently found after death in children who have died from debilitating disease of any kind, although no pulmonary symptoms may have been noticed.

For the most part the areas of collapse are small and are definitely related to areas of consolidation or to prominences on the inner wall of the thorax. On occasion, however, a whole lobe, or the greater part of a lobe, or even a whole lung, may undergo collapse (massive collapse).¹ This type of collapse has been observed most frequently in connection with foreign bodies in the bronchi, diphtheria, and after operations on the chest or abdomen, but it may occur apparently spontaneously or in the presence of a mild pulmonary or bronchial infection.

Causes.—The special tendency to the occurrence of collapse in young children is explained partly by the soft and yielding chest-walls, the weak respiratory muscles, and the fact that coughing, in them, is mainly a reflex act and is not aided, as in adults, by experienced voluntary efforts at expectoration. A smaller amount of bronchial secretion will therefore obstruct the free entrance of air into the vesicles; and narrowing of the upper air-passages, such as occurs from adenoids, is more apt to hinder the lung expansion.

Massive collapse, however, is probably not caused by simple obstruction with mucus, nor by paralysis of the diaphragm or chest-wall, as has been suggested. Spasm of the bronchi and of the whole musculature of the lung plays an important part. As Woodburn Morison² says, "it is a reflex disturbance of the vagus and sympathetic innervation of the lungs." Through over-action of the sympathetic, brought about by many causes, there results "dilatation of bronchi with cessation of the wave-like movement and a consequent accumulation of mucus. The balance between the sympathetic and vagus may be quickly

¹ L. Findlay, *Proc. Roy. Soc. Med.*, 1932, xxv. (Child. Sect. 17).

² J. W. Woodburn Morison, *Brit. Med. Journ.*, 1930, vol. ii., 237.

restored and the condition rapidly clear up with return of cough and the expectoration of mucus. On the other hand, as the over-action of the sympathetic passes off, the vagus may reassert itself too strongly and cause a sudden spasm of the musculature, involving the whole lung, or a lobe of the lung, or even, in rare cases, both lungs. It is not necessary to conceive of the spasm persisting any great length of time ;

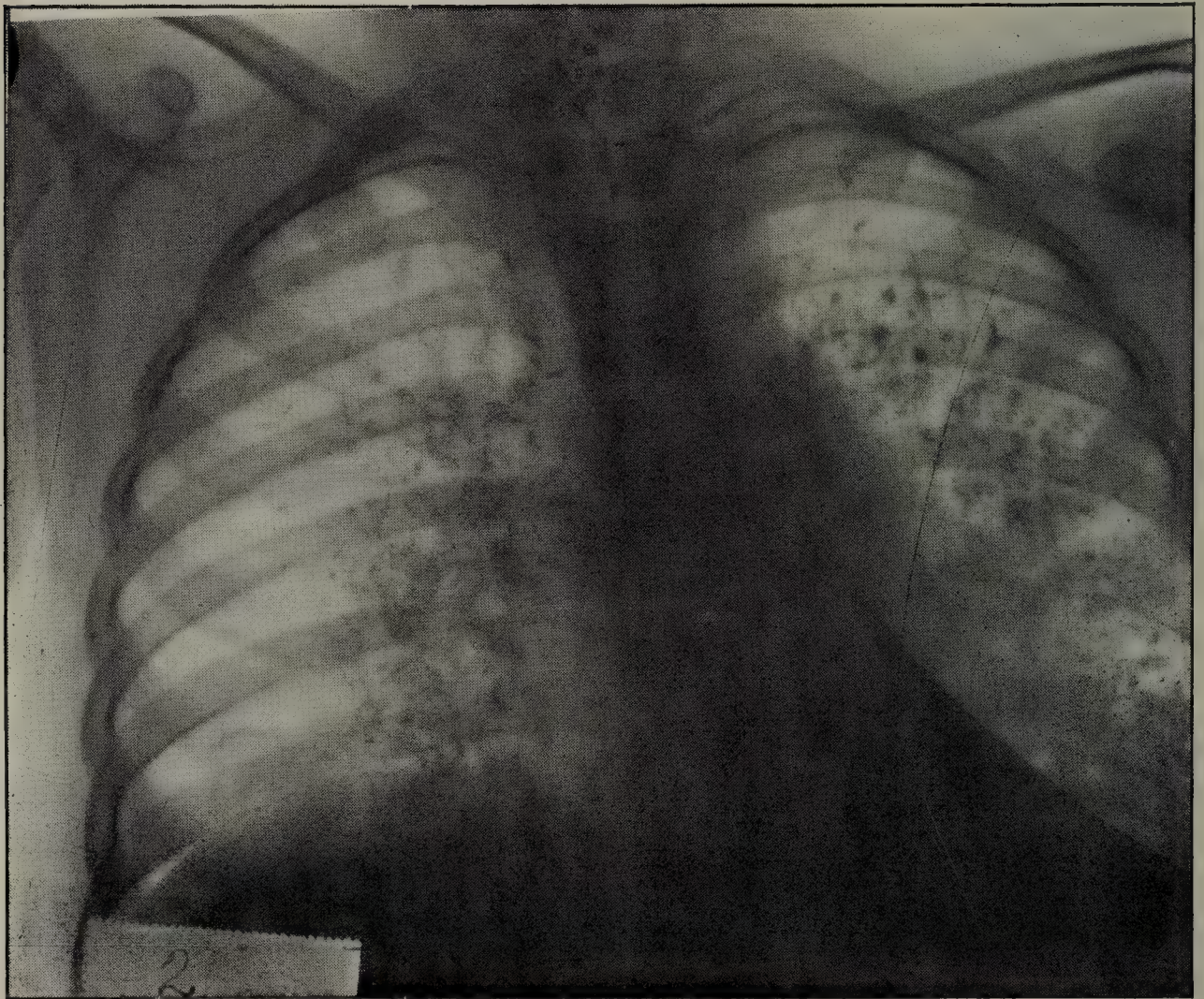


FIG. 196.—Skiagram taken just before inhalation of carbon dioxide. Note the dense triangular shadow due to collapse of lower lobe of left lung. The mottling in this and the following picture is the result of injection of lipiodol some days previously.

it not only empties the lung tissue more or less of its air content, but when the walls of the alveoli and terminal bronchioles are brought into apposition the molecular adhesion effectively prevents the re-entry of air."

Symptoms.—The symptoms of collapse vary according to the amount of lung involved. If a large area is suddenly affected in this way, the respiration becomes very rapid, rising perhaps to 70 or 90 in the minute, the *alæ nasi* work violently, cyanosis appears, and the child is greatly distressed. The cough is less loud and harsh than previously, and the child

shows signs of exhaustion. The temperature does not rise as in pneumonia, and may even fall.

The physical signs are variable. If the area of collapse is large the corresponding portion of the chest-wall is immobile. There is usually dullness to percussion, with feeble breath-sounds, though the respiratory murmur may be intensely amphoric in character and whispered pectoriloquy audible. The heart, as a

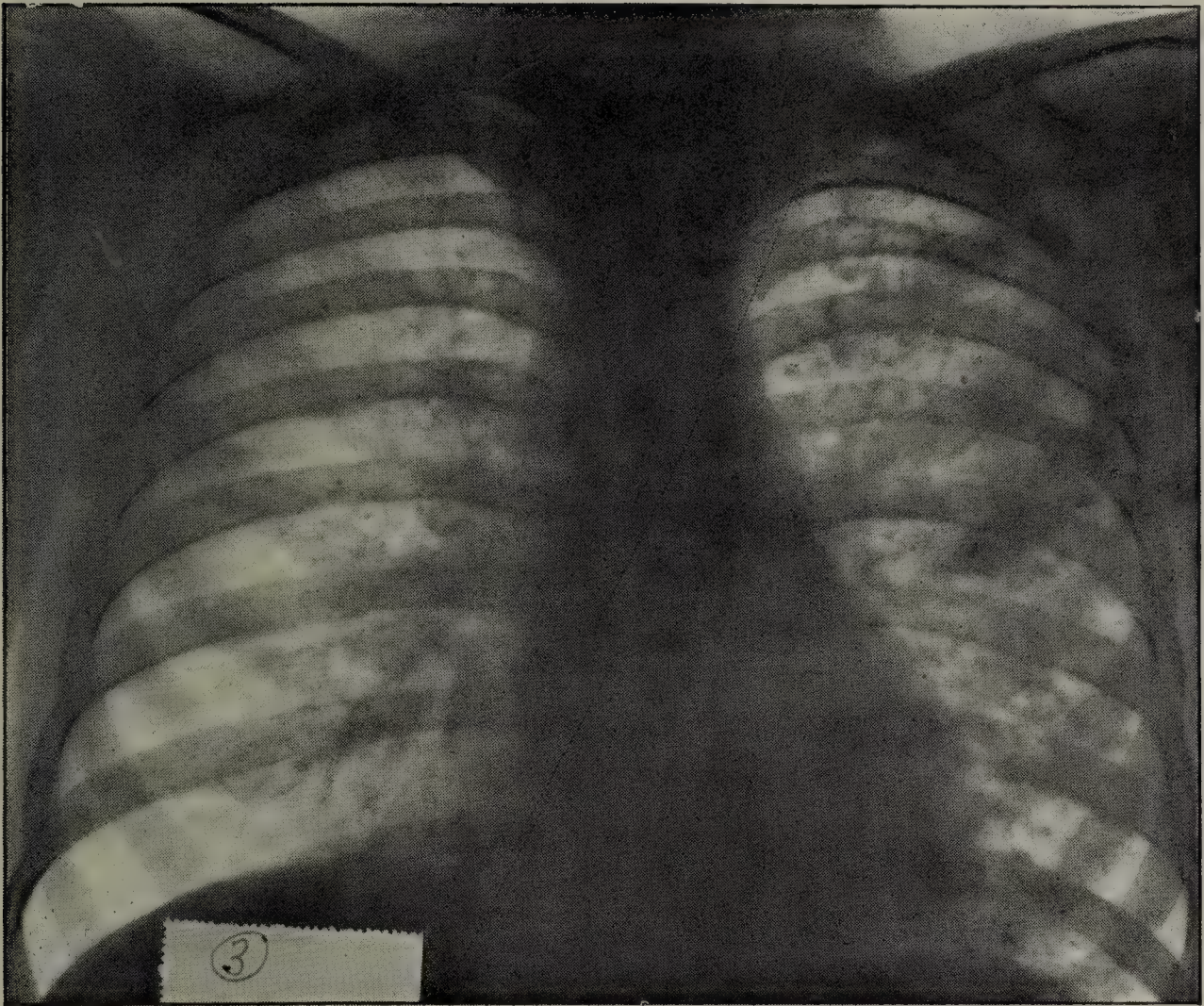


FIG. 197.—Skiagram taken two days after inhalation of carbon dioxide, showing disappearance of area of collapse.

rule, is displaced to the affected side. The collapsed portion of lung has a much diminished translucence to the X-rays and pictures suggesting fibrosis, pneumonic consolidation or fluid may be obtained. In Fig. 196 is shown the X-ray appearance in a case of collapse of the left lower lobe. A shadow similar to this may result from fibrosis (Fig. 198, p. 632) and is said to occur in mediastinitis or a mediastinal pleurisy.

From the physical signs alone it is impossible to decide as to the nature of the lesion, but the mode of onset, the absence of fever, and especially the response to attempts at reinflation will usually settle the diagnosis.

Treatment consists in attempting to reinflate the lung. This may be brought about by causing the patient to take sudden and full inspirations, which is best effected by the inhalation of 10 per cent. CO_2 in oxygen, but the sudden application of cold to the chest, or by rubbing with a stimulat-

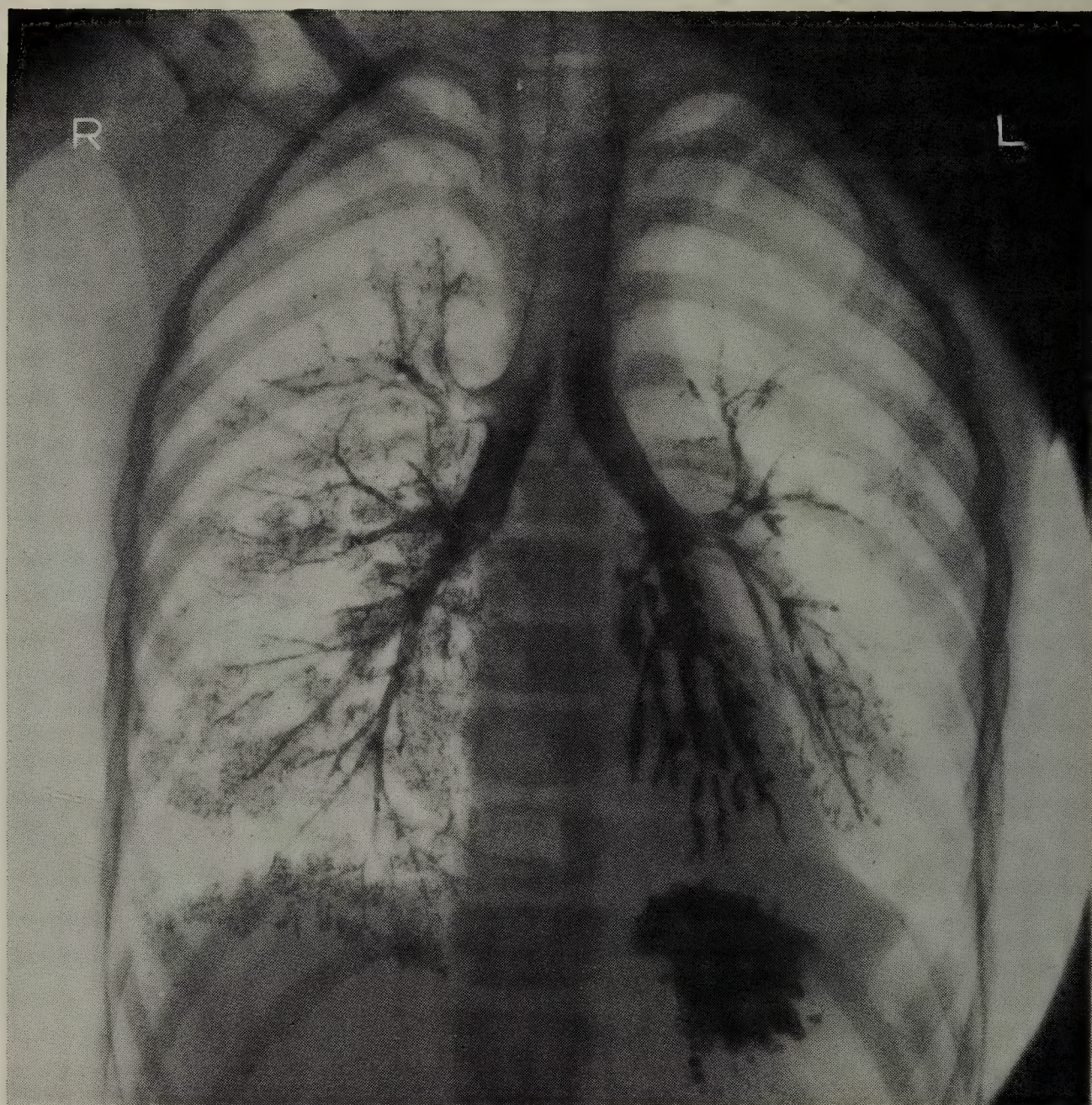


FIG. 198.—Skiagram after lipiodol injection in case of Bronchiectasis. Note the dilated bronchi in sclerosed portion of lung. Compare with Fig. 196.

ing liniment, or forcible compression and sudden relaxation of the chest-wall, may also have the desired effect. The rapid disappearance of the radiological appearances, as also occurred in the case of the physical signs, are shown in the X-ray photographs (Figs. 196 and 197, pp. 630 and 631) of massive collapse in a boy aged seven years.

Pleurisy.

Dry pleurisy, and pleurisy with serous effusion, apart from pneumonia or tuberculosis, are comparatively rare in early infancy (Fig. 199). In older children they are more common, though less so than in adults. Serous pleurisy is, as in the adult, usually of tuberculous origin in childhood. It is very doubtful if pleurisy is ever due to the rheumatic infection. The predisposing causes are those met with in adult life, and generally there is no important difference in the physical signs in children, except that often no friction is heard. The general symptoms, even with a large effusion, are often surprisingly slight.

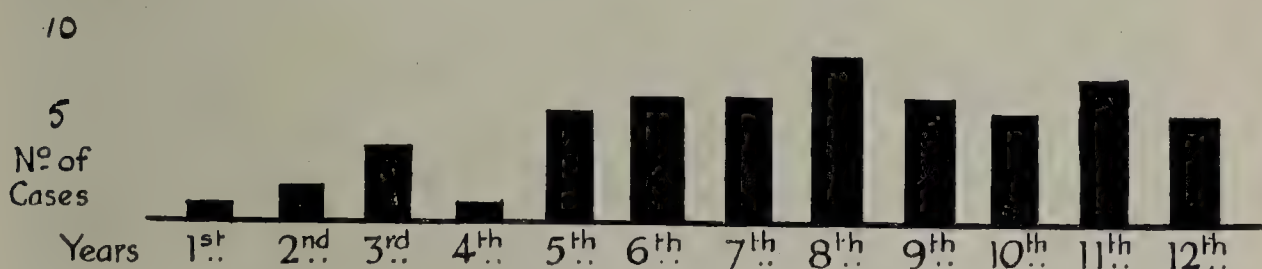


FIG. 199.—Age Incidence during childhood of Pleurisy with Effusion.

The **prognosis** is generally good in older and less weakly children, but the process of recovery may be very slow. Recovery is almost the rule if the general conditions are favourable and they are energetically treated. In childhood there is a much less tendency for pulmonary tuberculosis to develop later: perhaps the commonest sequela is spinal caries.¹

The **treatment** does not differ from that in the adult.

Pneumothorax.

Pneumothorax is not common in childhood, and is rare in babies. It is met with under various conditions—(1) Pyo-pneumothorax, where the gas present arises from the decomposition of a septic pleural exudate, is sometimes found, though much less frequently than in adults. (2) Following exploratory puncture, in cases where there are pleural adhesions or partial consolidation of the lung (Fig. 200). (3) Most commonly from the passage of air from the lung into the pleura, as the result of

¹ S. Graham, *Glasg. Med. Journ.*, 1925, civ., 1.

violent coughing. This may be due to rupture of a tuberculous cavity or emphysematous patch; but it sometimes occurs when the lung is apparently healthy, owing to sudden increase of the intrapulmonary pressure during very severe coughing—as in

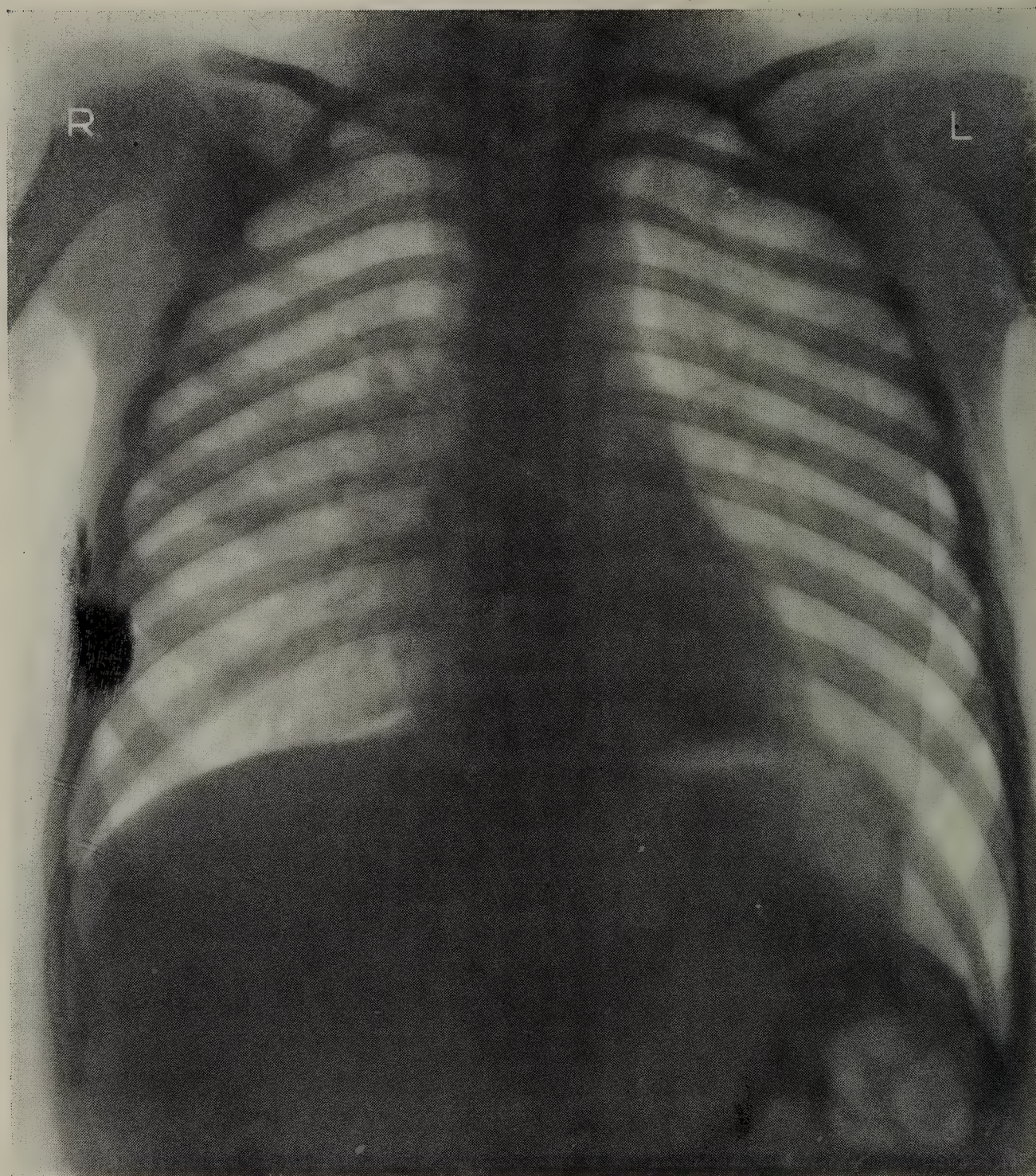


FIG. 200.—Skiagram of Pneumothorax developing after exploration of chest.
(Boy aged 3 years.)

whooping-cough or measles (see Subcutaneous Emphysema, p. 383).

In pyo-pneumothorax, incision and drainage are generally called for. In the cases due to violent coughing, no active measures are indicated beyond soothing the cough. We have seen several such cases end in apparently complete recovery.

Empyema.

Empyema is much commoner in children than in adults and may occur even in the first week or two of life. It is often more insidious in its onset and more obscure in its symptoms than in later life. In children also it is less frequently tuberculous in origin, and more often met with in a localised form.

Causes.—The general condition of the child's nutrition is of great importance in determining the onset of empyema. It occurs frequently among the ill-nourished children of city slums under circumstances in which it is rare among the upper classes or in country children. It is often a sequel to scarlet fever, measles, or some other infectious disease, but it is most frequently met in association with pneumonia, and especially lobar pneumonia. Of the examples of pneumonia under the care of one of us (L. F.) in R.H.S.C., Glasgow, empyema occurred in 18.4 per cent. of those of the lobar and in 5.2 per cent. of those of the broncho-pneumonic variety, while McNeil¹ and his co-workers in Edinburgh found that, of 58 cases of empyema in which the nature of the accompanying pneumonia could be definitely decided, this was of the lobar or alveolar type in 77 per cent. It is important to appreciate, too, that empyema may occur either as a true complication, *i.e.*, develop during the course of the pneumonia, when it is spoken of as *syn-pneumonic*, or it may only develop as a sequel after the pneumonic inflammation has terminated, when it is called *meta-pneumonic*.² This differentiation of time of onset is not only important from the points of view of diagnosis and prognosis, but also from that of treatment.

In children far the commonest cause of empyema is the pneumococcus. Other pyogenic organisms, especially streptococci, are often to blame, and in these the prognosis is distinctly better. The tubercle bacillus is much less often found.

Symptoms.—The symptoms vary in different cases. When the condition develops subsequent to pneumonia (meta-pneumonic) the return of fever after a varying period, with cough, malaise, loss of appetite, pallor, and sweating, quickly suggest the possibility of its occurrence. It is otherwise,

¹ C. McNeil, A. R. MacGregor, and W. A. Alexander, *Arch. Dis. Child.*, 1929, iv., 18.

² H. C. Cameron and A. A. Osman, *Lancet*, 1923, i., 1097.

however, when the purulent exudate develops *pari passu* with pneumonia (syn-pneumonic), since the exacerbation of fever or its continuance, especially when this is of the broncho-pneumonic type, may be looked upon simply as a persistence of the primary disease. A septic type of fever and profuse sweating should always raise the question of such a complication. It must also be remembered that empyema may be present when there is a complete absence of fever.

Physical Signs.—On examining the chest, if the collection of pus is small and localised, there may be only a patch of dullness with diminished breath-sounds and defective resonance. This is oftenest found near the angle of the scapula, but it may be situated in any region of the chest, and occasionally occurs over the apex in front, with resonant lung below it. If the pus is present in large amount, and is free in the pleural cavity, as is usually the case where the illness is of recent origin and has come on acutely, there is generally some bulging of the affected side, with diminished movement. The heart may have been displaced by the pressure of the fluid. In chronic cases the affected side is sometimes shrunk, owing to the pulmonary collapse which is present, and the heart is drawn towards the affected side. On palpation, auscultation, and percussion, there are the usual signs of fluid. The temperature may be raised, but it is frequently quite normal; more or less marked leucocytosis is always present. If the empyema has lasted for any length of time, there is generally clubbing of the finger-ends. The occurrence of empyema on both sides of the chest is not very uncommon. In obstinate cases of empyema or abscess of the lung due to a foreign body, an X-ray examination may be of great assistance in revealing the real cause of the symptoms.

The only certain way of making a diagnosis, however, is to use an exploring syringe, and this we should *invariably* do if in doubt. If one puncture fails to discover pus where there is reason to expect its presence, the process should always be repeated (frequently if necessary) either at one sitting or at intervals of a day or two. With ordinary aseptic precautions, no harm results from the puncture. The only accidents we have seen are severe hæmoptysis and cutaneous emphysema. The former only occurred once and the latter in some half-dozen cases. When emphysema takes place, it begins round the site of

the puncture. It occurs when the needle passes through air-containing lung tissue which is adherent to the chest-wall, and it merely occasions a slight rise of temperature for a few days. Exploring for pus requires a larger syringe than is used for subcutaneous injections, and also a considerably larger needle than the ordinary hypodermic one. If the pus is thick, as in many chronic cases, an ordinary hypodermic syringe may entirely fail to reveal its presence.

Prognosis. — Empyema, however treated, is a serious disease during early childhood. Irrespective of the type of accompanying pneumonia, McNeil¹ found that the mortality rate was 75 per cent. during the first two years of life, 31·5 per cent. from two to five years, and 8·5 per cent. from five to twelve years. The experience in the West of Scotland has been hardly so satisfactory. Of the cases admitted to R.H.S.C., Glasgow, the mortality varied between 77 and 60 per cent. during the first five years of life and from five to twelve years of age was 14 per cent.

As already mentioned, prognosis depends on whether the effusion is syn-pneumonic or meta-pneumonic in type. McNeil puts the death rate for all ages of the latter at 9 per cent. and of the former type at 71 per cent. At the R.H.S.C., Glasgow, the mortality from empyema following broncho-pneumonia was always higher than from that due to lobar pneumonia, irrespective of whether it was syn- or meta-pneumonic in onset. In empyema due to broncho-pneumonia the death rate in the syn-pneumonic variety was 86 per cent. and in the meta-pneumonic 61·2 per cent., while the corresponding figures for the condition due to lobar pneumonia were 50 per cent. and 7·6 per cent. respectively. There is no doubt that the high death rate from empyema during the early years of life is due to the fact that the primary lesion is usually, if not invariably, of a broncho-pneumonic nature (to the duration of which there is no time limit, as is the case with lobar pneumonia) and that persistent activity of the pulmonary lesion is a very large factor in contributing to the fatal termination.

Treatment.—The treatment of empyema consists, of course, in the speedy removal of the pus, but the particular method by which this is accomplished depends on the age of the child, the

¹ C. McNeil, A. R. MacGregor, and W. A. Alexander, *Arch. Dis. Child.*, 1929, iv., 270.

acuteness of the illness, and whether or not there is any active pneumonic mischief present.

If the patient is an infant, acutely ill, and there is evidence or suspicion that the pneumonic condition is still active, aspiration is probably the wise proceeding. This may be repeated on several successive occasions, and although the pus always reaccumulates, the child is rendered less poisoned, the breathing is relieved, time is given for the active pneumonic condition to subside, and in consequence any subsequent operation is rendered more safe.

Occasionally a single aspiration may suffice, but in our experience, if this has been unsuccessful, incision of the pleura has ultimately been found necessary. If the ribs are fairly wide apart, a simple incision, and subsequent draining with an india-rubber tube, will probably prove the best treatment, especially if the child is feeble. If the ribs are very close together, however, or if a simple incision has been made and the drainage from it is not satisfactory, it will be well to consider the advisability of resecting a portion of a rib. This has the disadvantage of generally necessitating the use of a general anæsthetic (which is more dangerous in these cases than in almost any others in childhood), but it has compensating advantages. It allows the masses of semi-solid purulent lymph, which are often found, to be more completely evacuated; it ensures freer drainage; and last but not least, it makes the dressing a less painful process than in cases of simple incision when the tube fits tightly between the ribs. Washing out the cavity is never advisable except perhaps in cases where the pus is offensive in character, and these are very rare in childhood.

If the operation is done in time and the cavity drains well, the result is generally most satisfactory; and, in favourable cases, the tube may be removed in two or three weeks. Any rise of temperature generally signifies some interference with the drainage, and may require the use of a larger or a second tube.

McNeil,¹ instead of having recourse to incision because several aspirations are unsuccessful, recommends the introduction of a special short silver cannula of wide bore, which is retained *in situ* and through which the cavity is allowed to drain and is aspirated if necessary once or twice or more times

¹ C. McNeil, *The Practitioner*, 1930, cxxv., 219.

daily; with this technique McNeil claims that the mortality has been reduced. In our hands this method did not prove very successful.

Occasionally a staphylococcus infection of the wound causes a return of the fever and an increased discharge. This may prove obstinate, but it can often be very satisfactorily treated by the use of an autogenous vaccine. Another common cause of rise of temperature during recovery from empyema is the occurrence of a pneumococcus otitis. Infections of the peritoneum and meninges are occasionally met with; and, in young children who are poorly nourished, a spread to the pericardium is not very rare. In cases where treatment has been unduly delayed, a considerable amount of collapse of the lung may persist, and this may lead in after-life to fibrosis and bronchiectasis.

Bronchitis.

Bronchial catarrh is extremely common in infants and young children, and is important, not only for this reason, but also because it is so liable in them to be complicated by pulmonary collapse and broncho-pneumonia.

Causes.—Cold and damp are common *exciting causes*, and also injudicious clothing and exposure. To prevent chills in examining infants, therefore, we should avoid exposing much of the surface of the body at a time, except in a warm room or in front of a good fire.

In many cases there are also important *predisposing causes* at work, and these have to be taken into account in the treatment. Rickets strongly favours the occurrence of bronchial as well as other forms of catarrh, and a protracted or recurring bronchitis may be due to this. In rickety cases the regulation of the diet and other antirachitic measures are of more importance in the treatment than cough mixtures. In other cases the recurrence of bronchitis is due to the presence of "lithæmia," and sometimes the attacks may turn out to be of the nature of asthma or acidosis.

Even in children who are not rickety, teething seems sometimes to predispose to bronchitis, and the advent of each new group of teeth is accompanied by an attack. Another important predisposing cause of recurrent bronchial attacks is the presence of adenoid growths in the naso-

pharynx, and, as Lockhart Gibson and others pointed out long ago, the removal of these is often followed by comparative immunity from further recurrence of the bronchitis. Too much credit must not, however, be given any therapeutic measure, as bronchitis in childhood shows a remarkable tendency to ameliorate or disappear about the age of seven or eight years.

Symptoms.—The symptoms are much the same as in later life—a slight rise in temperature, a cough usually deep and loud, slight acceleration of the breathing, no dullness or bronchial breathing, loud sonorous ronchi and bubbling râles if the larger bronchi are affected, and sibilant and crackling sounds if the smaller tubes are involved. They vary in severity not only with the size of the bronchi affected and the stage of the attack, but also with the strength of the patient. In wasted and weakly infants the symptoms are less severe, although the real danger is much greater than in those who are robust. If the child's cough and cry are loud, the case is not yet a serious one.

Treatment.—In treating a baby with acute bronchitis there are two main indications. *First*, the strength must be sustained, and depressing influences guarded against. The room should be kept about 60° F., and be well-aired, although all direct draughts are to be avoided. The diet must be as nourishing and digestible as possible, and if the baby is refusing food, or if the case is a severe one, stimulants may be advisable—alcohol, ammonia, and strychnine.

Second, the patient must be assisted to get rid of the excessive bronchial secretion which constitutes an element of danger to him, by the secretion being rendered more fluid and the natural methods of its expulsion aided. This indication may be fulfilled in various ways—

(a) The air he breathes should be moistened by having a steam-kettle playing close to his bed, or by hanging up near it, from time to time, towels wrung out of hot water.

(b) Hot fomentations may be applied to the chest if the case is acute, or perhaps better, a mustard poultice or pack followed by a cotton-wool jacket; or a stimulant embrocation (Appendix E, Form. 31 and 32) may be used.

(c) In the early stages small doses of ipecacuanha wine should be given with an alkali. Later, such stimulant expectorants as carbonate of ammonia and squills are

indicated, and a sedative may sometimes be added with advantage (Appendix E, Form. 15 and 16).

(*d*) When, in a strong child, the secretion is copious and the patient is not vomiting spontaneously, an occasional emetic may give marked relief. If the baby can be easily made to vomit by tickling his fauces with a feather, this is better than giving an emetic.

(*e*) The clothes must be warm, and should not be so tight as to interfere with the full play of his chest in breathing and coughing.

When the attack is over, it is always most important to see that the child's health is thoroughly restored, and not to be contented with an incomplete convalescence. The use of cod-liver oil and tonics and a change of air are generally desirable.

Bronchiectasis.

Dilatation of the smaller bronchi is probably not uncommon in broncho-pneumonia, at least it is a frequent post-mortem finding. This is undoubtedly due to the bronchi being implicated in the inflammatory process, but in all probability it is usually recovered from, considering the frequency of broncho-pneumonia and the rarity of bronchiectasis in after-life. It would seem that it is only when definite fibrotic changes in the lung supervene that there is any danger of permanent bronchiectasis developing. In fact, bronchiectasis as a clinical entity and pulmonary fibrosis are synonymous terms.¹ McNeil and his colleagues,² from histological studies of different stages of the disease, incline to the view that in certain cases at least the primary change is in the bronchial wall. In their opinion this has undergone destruction and has been replaced by a new fibrous structure which, being devoid of muscle, elastic tissue, and cartilage, tends to dilate. The pulmonary fibrosis in these cases seemed to be secondary, although they admit it might further increase the bronchial dilatation. Pinchin and Morlock³ have recently suggested that in some examples the primary lesion may be pulmonary

¹ L. Findlay and S. Graham, *Arch. Dis. Child.*, 1927, ii., 71.

² C. McNeil, A. R. MacGregor, and W. A. Alexander, *Arch. Dis. Child.*, 1929, iv., 174.

³ A. J. Scott Pinchin and H. V. Morlock, *Brit. Med. Journ.*, 1930, i., 12.

atelectasis which undergoes fibrosis and in this way produces the bronchiectasis.

In the vast majority of cases the condition dates from a broncho-pneumonia, often secondary to measles or whooping-cough, but it may be a sequel to a typical lobar pneumonia, a pleurisy with effusion, or any lung lesion which may lead to pulmonary fibrosis and an anchoring of the lung. In a few instances it seems to develop spontaneously, although recently it has been suggested that such examples may be of congenital origin. It is stated by some writers to be a common sequel to suppuration of the accessory nasal sinuses, but this has not been our experience.

One of the most striking features of this disease is its distribution. It tends to be limited to one lung and especially to one lobe of a lung, to involve the base of the lung rather than the apex, and to attack the left lung much more frequently than the right. This distribution is in marked contrast to pulmonary tuberculosis, which shows a predilection for the apex of the lung. The dilatation of the bronchi may only affect their terminations and give rise to a saccular bronchiectasis or it may involve the bronchi in a considerable extent of their length, producing a tubular bronchiectasis. These various points are well illustrated in Figs. 203 to 207.

Symptoms.¹—In the first place it must be appreciated that the condition may be present in a marked degree and yet give rise to no symptoms, and may only be brought to light through the development of a cerebral abscess or it may be an accidental finding in the post-mortem room. In some cases symptoms are present without any signs and in others signs without any symptoms, but very often both signs and symptoms are present.

When symptoms are present these are a characteristically spasmodic cough, coming in bouts like whooping-cough and often precipitated by exertion or on assuming certain positions. Cough is indeed the most constant of all symptoms. Frequently the cough is accompanied by a profuse purulent expectoration which may have, but rarely has, a fetid odour. The presence of sputum is related more to the duration of the illness than to the severity of the condition and in this respect contrasts with clubbing of the fingers.

The physical signs vary with the seat and extent of the

¹ L. Findlay and S. Graham, *Arch. Dis. Child.*, 1927, ii., 71.

lesion, which in the majority of instances is situated in the left lower lobe. The right lower lobe is much less frequently involved. The whole lung may be fibrosed and the seat of generalised bronchiectasis, but the upper lobes never seem to suffer alone. Commensurate with the extent of the lesion is retraction of the chest-wall and dullness to percussion. On auscultation the respiratory murmur is tubular, or intensely amphoric, and the whispered voice is well heard. If the bronchi are filled with secretion these two auscultatory phenomena may not be heard, and hence, before deciding as to their absence, the child should be inverted and made to cough (posturing) so as to attempt to clear the air-passages.

Clubbing of the fingers and toes is often present, and, as previously mentioned, is more related to the extent of the mischief than to its duration. There may be a degree of cyanosis. Growth and nutrition in the more marked examples suffer, and hence the children are under weight and height.

The temperature may be normal but is subject to periodic rises, probably in consequence of attacks of broncho-pneumonia, which indeed is a not uncommon cause of a fatal termination. Abscess of the brain may also develop as a complication.

Diagnosis.—During childhood an unresolved pneumonia with fibrosis and bronchiectasis is the commonest cause of a chronic cough with purulent expectoration. Unfortunately this condition is frequently diagnosed as pulmonary tuberculosis, but this, as we will point out later (p. 974), is at this period of life almost invariably an acute disease. Thus the longer the duration of the cough and spit the less likely are they to be dependent on a tuberculous lesion and the more likely to be due to bronchiectasis. It may be taken as a safe rule never to diagnose chronic pulmonary tuberculosis in the child without the presence of tubercle bacilli in the sputum.

Although the symptoms and signs may be characteristic, it must be remembered that the condition may exist in the absence of either, and also that typical amphoric breathing and whispered pectoriloquy may be present in an exquisite degree when no bronchiectasis exists, as, *e.g.*, in the case of a slowly resolving pneumonia. The only certain method of detecting bronchiectasis, as also of learning of its true extent, is by radiology after the intratracheal injection of lipiodol. By this method the conformation of the whole bronchial tree is discerned

(Figs. 201 and 202). In our experience the best results are obtained in children by the introduction of the lipiodol

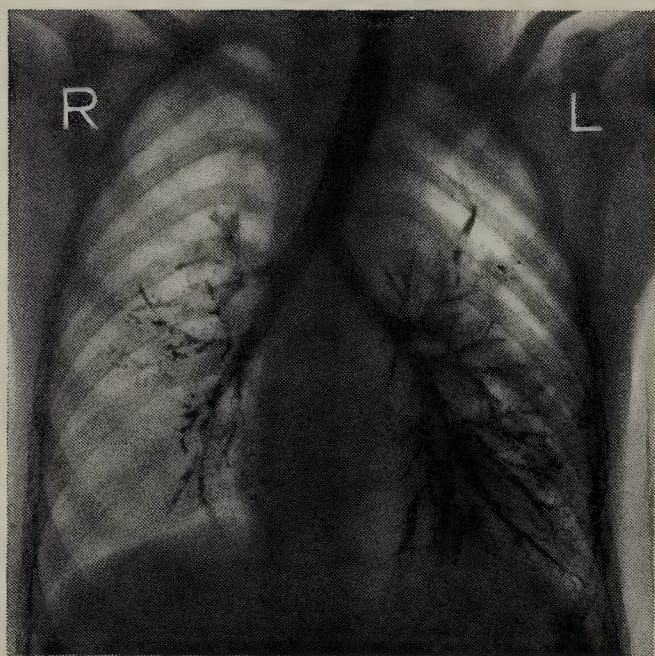


FIG. 201.—Skiagram after lipiodol injection in case of slowly resolving Pneumonia, showing normal bronchial tree. (Girl aged 9 years.)

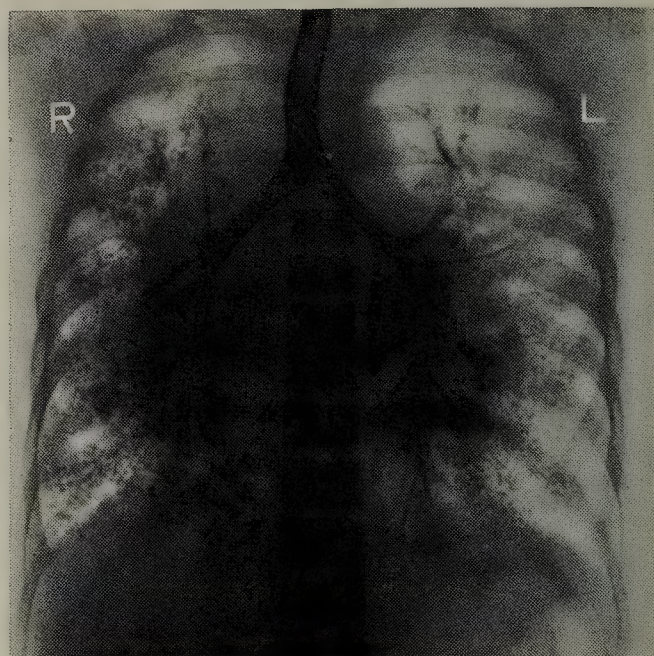
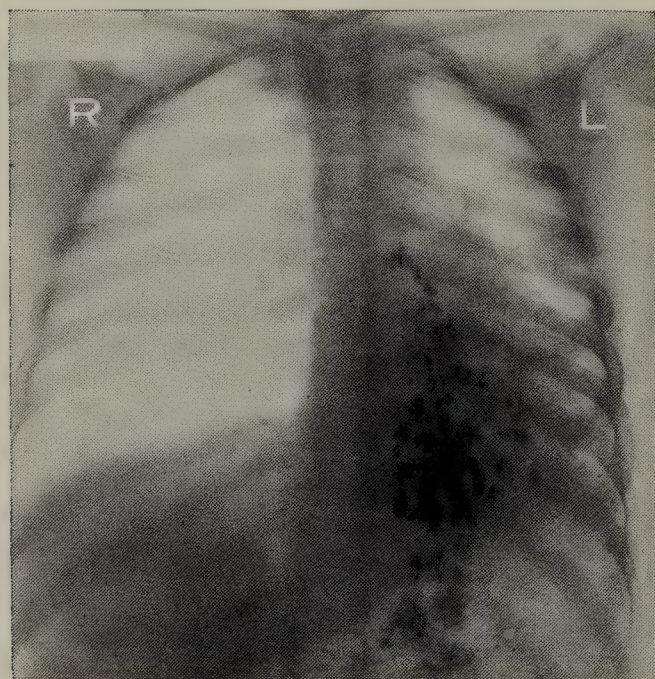
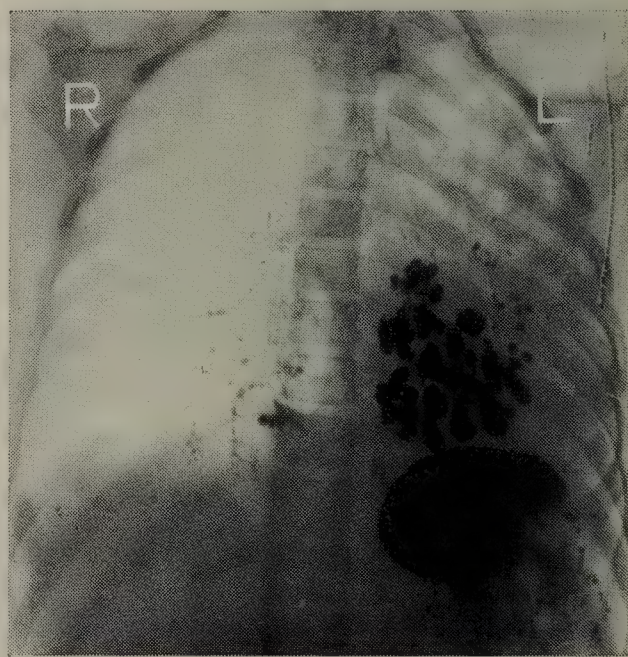


FIG. 202.—Skiagram after lipiodol injection in case of slowly resolving Pneumonia without Bronchiectasis, showing powdering effect due to opaque substance in alveoli. (Girl aged 4 years.)



6.12.24

FIG. 203.—Skiagram after lipiodol in Saccular Bronchiectasis limited to left base. (Same case as Figs. 204 and 205.) (Girl aged 7 years.)



10.1.27

FIG. 204.—Skiagram after lipiodol injection in case of Saccular Bronchiectasis in left lower lobe. (From same case as Figs. 203 and 205.)

through the cricothyroid membrane under general anæsthesia and when fairly large quantities (20 to 30 c.c.) of the opaque oil are employed. When no coughing occurs during the operation the bronchi are sharply delimited, and it is easily seen

whether they gradually taper towards their extremities (Fig. 201) or are dilated. If, however, coughing has occurred before the skiagram is taken, the lipiodol may have been forced into the alveoli, giving the appearance of powdering and obscuring the outlines of the bronchi (Fig. 202). The presence of lipiodol in the alveoli usually indicates that that portion of the lung is healthy.

Prognosis.—Contrary to the opinion expressed by Thursfield and Paterson,¹ in our experience the condition gradually tends to get worse although it is compatible with long life. In fact most of the cases met with in the adult date from childhood.² If, however, the condition is very slight and of short duration it may recover completely; this, however, is rare. In spite of the fact that the lesion as demonstrated by radiograms is steadily progressive there is a tendency for the cough and spit to be less troublesome during later childhood and in some cases they entirely disappear.³ (Figs. 203 to 205.)

The **treatment** of bronchiectasis is most unsatisfactory. We have never seen any permanent benefit from continuous



26.4.30

FIG. 205.—Skiagram after lipiodol, showing extensive Tubular Bronchiectasis of left lung. (Same case as Figs. 203 and 204.)

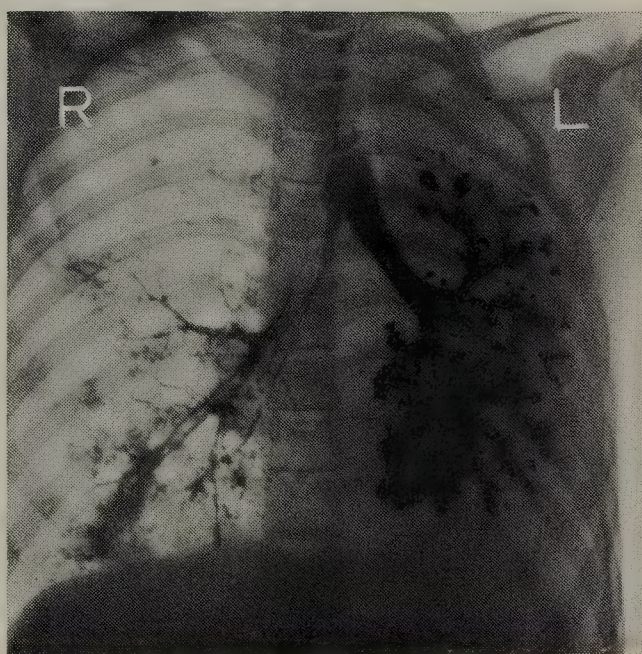


FIG. 206.—Skiagram after lipiodol injection of extreme degree of Tubular and Saccular Bronchiectasis. (Girl aged 11 years.)

¹ J. H. Thursfield and D. Paterson, *Proc. Roy. Soc. Med.*, 1926, vol. xx. (Sect. Dis. Child., 1).

² A. Clark, W. J. Hadley, and A. Chaplin, *Fibroid Disease of the Lung*, London, 1894.

³ L. Findlay and S. Graham, *Arch. Dis. Child.*, 1931, vi., 1.

inhalations of creosote or other preparations, the use of the inclined plane or other method for the evacuation of the secretion, vaccines, artificial pneumothorax or oleothorax. Chevalier Jackson records good results from daily emptying

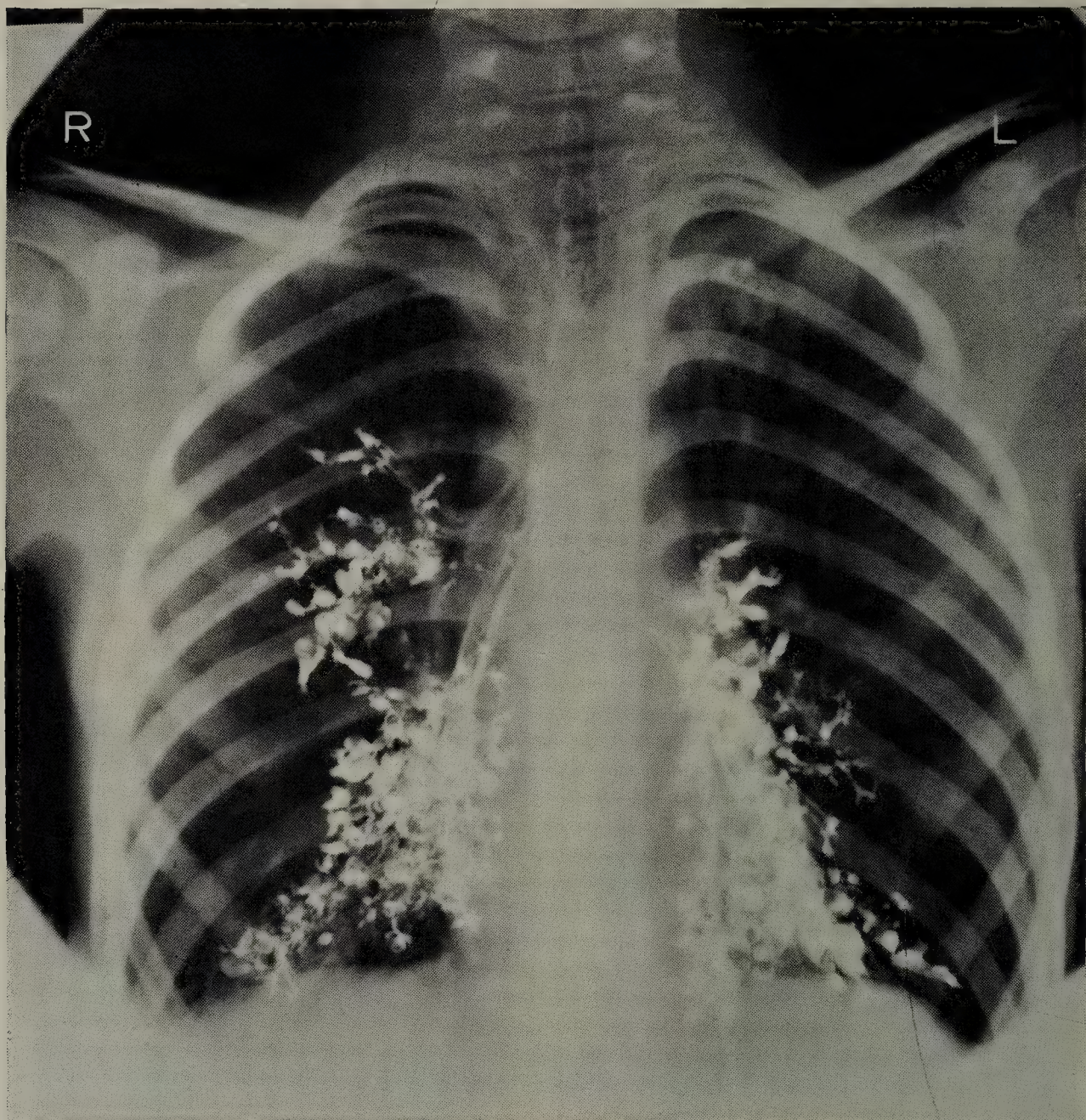


FIG. 207.—Skiagram after lipiodol injection in example of Sacculated Bronchiectasis affecting both bases. (Boy of 7 years.)

and washing out of the cavities. We have never seen any benefit result from the lipiodol injections.

Surgery would seem to hold out the greatest prospects of success, but the particular technique for excision of the diseased portion of the lung has not as yet been evolved. Excision of the whole lung has usually been fatal. Avulsion of the phrenic nerve, previously so much lauded, has fallen into disfavour.

Asthma.

Age of Onset.—The accompanying chart (Fig. 208) gives a graphic representation of the age at onset of the symptoms in one hundred cases of asthma in children under fourteen years, so far as this could be ascertained from the parents. It certainly puts the beginning of the disease somewhat later than it really was in many of them, for the early symptoms often pass unrecognised. It shows in a striking way to what an extent asthma is to be regarded as a disease of early childhood, and therefore emphasises the importance of always being on the lookout for its earliest manifestations. We see from it that the disease frequently sets in during the first twelve months (in two instances it began as early as the fourth and sixth weeks of life); and that, in most of the cases, symptoms of its presence were noted before the end of the fourth year. Comparatively few of them began in later childhood.

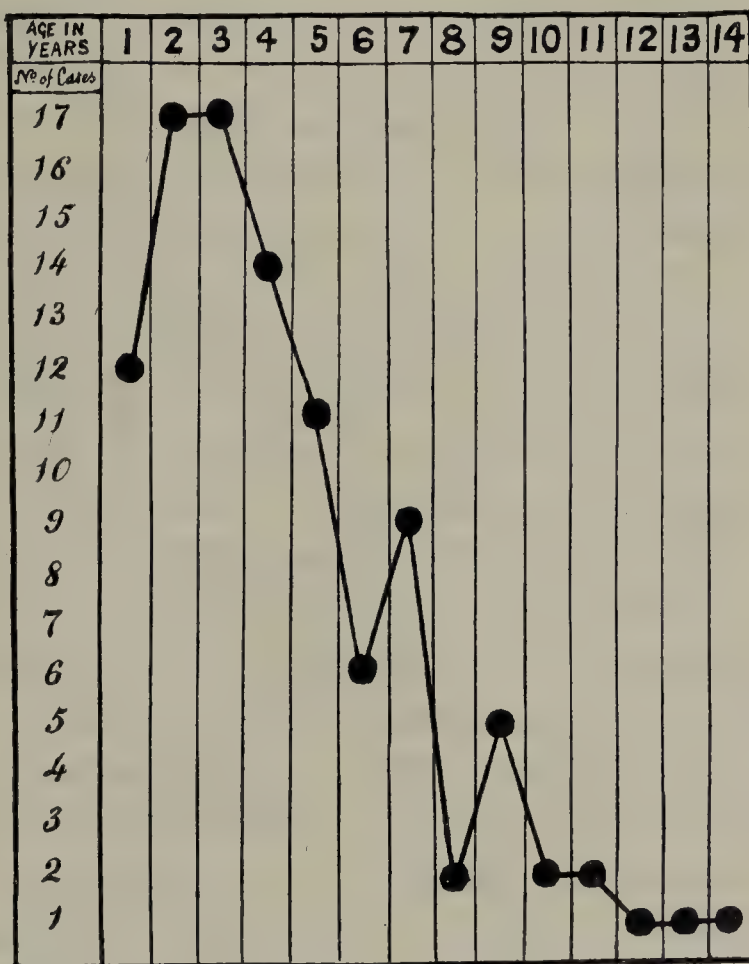


FIG. 208.—Age at onset of Asthma in 100 cases.

Sex.—Asthma is commoner in boys than in girls. In these hundred cases the ratio was exactly 3 : 2.

Family History.—The health of the child's immediate relatives may be interesting; for we often get a history of several of them having suffered from asthma, hay-fever, eczema, urticaria, or gout, or having had idiosyncrasies for some common forms of food, such as eggs, fish, the flesh of fowls, or oatmeal. Asthma is much commoner in well-to-do families than among hospital patients.

Accompanying Diseases and Complications.—The patients themselves, also, have not uncommonly suffered, and may still be suffering, from eczema or some other skin disease, from

nervous affections, from nasopharyngeal obstruction, or from an organic chest affection, or a digestive disturbance. Thus, in the one hundred cases, twenty-six had a history of eczema (usually in infancy and sometimes later), eight of urticaria, two of ichthyosis, one of psoriasis, five had enuresis, one of them also fæcal incontinence, two had had fits, one was suffering from habit-spasm, two of the older cases had hay-fever, and two suffered from abnormal obesity. In at least seventeen of the cases there were, or had been, enlarged tonsils and adenoids, in two there was distinct evidence of enlargement of the bronchial glands, in one of pleural adhesions, and in two of chronic pulmonary disease. In many of the cases there was emphysema, and, in some, a degree of cardiac dilatation. In several, idiosyncrasies for eggs and other foods had been noticed.

Some of these concomitant diseases and symptoms, such as the cutaneous and digestive disturbances and the food idiosyncrasies, are of special interest as indicating the frequent dependence of the bronchial spasm on anaphylaxis; some show merely the generally irritable state of the nervous system, while others, like the nasopharyngeal obstruction and the intra-thoracic lesions, probably have a causal connection with the asthma. The emphysema and the dilatation of the heart are obviously secondary to the respiratory distress.

Etiology.—The causation of asthma is supposed to be closely connected with a process of anaphylaxis or allergy. The patients, it is said, are, temporarily or continuously, in a state of sensitivity to some form of protein. In a large proportion of the cases the protein in question is contained in some special animal or vegetable food, for which the patient has an idiosyncrasy, and is absorbed from the alimentary canal. In others the offending substance is inhaled in the form of the dandriff, hair, or feathers of some animal, or the pollen of common grasses or flowers; and it may also be derived from pathogenic micro-organisms growing in some part of the body, such as the nose, throat, or intestine.

In recent years it has been found possible to ascertain which protein is to blame by means of a simple skin test. An abrasion, such as is used in Pirquet's tuberculin test, or a small cut, $\frac{1}{8}$ in. in length, and not deep enough to draw blood, is made on the flexor surface of the forearm. On this is placed a single drop of a solution of the protein to be tested, in a decinormal

solution of sodium hydroxide. Another similar abrasion or cut is made as a control, and to it only the sodium hydroxide solution is applied. After half an hour the solution of the protein is washed off, and the reaction noted. If it is positive, a raised white area or an urticarial wheal, at least 0.5 cm. in diameter, is found surrounding the abrasion. The most important forms of protein to try in this way are those of common foods, such as eggs, milk, various meats, cereals, and potato, and those of horse-hair, feathers, pollen from the commoner grasses, varieties of staphylococci and streptococci, pneumococci of types I and IV, and the diphtheroid bacillus. In cases of an idiosyncrasy for eggs, the inunction of raw white of egg into the unabraded skin may give a very definite reaction in the form of urticarial wheals.

Exciting Causes.—In children who are predisposed to asthma the attacks are frequently brought on by catarrh of the upper air-passages, or by teething, sometimes, though probably not so often as in adults, by dyspepsia, and often by taking eggs or some other special food. In one of the older cases excitement, laughter, and loud talking always set up an attack. Dust and emanations from the skin of certain animals such as the horse or cat may start one.

Predisposing Causes.—Climate is sometimes important in connection with the causation of the symptoms; and, as in adults, the place which suits one child best may be bad for another.

The presence of emphysema seems to have a strongly predisposing influence; and this probably accounts for the frequency with which a first attack occurs after whooping-cough or some other severe respiratory disorder.

Symptoms.—When asthma begins in infancy or early childhood, its earliest manifestations are often obscure, largely because they are associated with so much catarrh. The attack generally assumes the characters of acute bronchitis, but there is frequently a good deal of paroxysmal sneezing with it, its onset is curiously sudden, and there is apt to be wheezing, as well as urgent coughing. Usually some rise of temperature occurs, and the breathing may be so rapid (40 to 100) that broncho-pneumonia is often diagnosed. This diagnosis may be supported by the severity of the dyspnoea and by cyanosis and prostration. These urgent symptoms often pass off rapidly

within twenty-four to forty-eight hours, and this, along with their recurrence on subsequent occasions without any definite evidence of consolidation developing, clears up the diagnosis. The attacks vary, of course, in frequency and in severity. During the intervals minor paroxysms may occur, but the children are often surprisingly well.

As the child grows older the catarrhal symptoms become less and less prominent and the temperature less often rises, while the evidence of bronchial spasm is more evident. By the time the child is five or six years old the asthmatic character of the attack is unmistakable.

In cases in which the disease begins in later childhood the attacks generally resemble those in adults from the first, and there is little difficulty in recognising their nature.

The **prognosis** is usually more favourable in those cases in which the disease begins in early childhood; but the cases which are set up by odours, pollen, and special foods are often obstinate. When the symptoms are secondary to emphysema, or when the attacks have been allowed to go on unchecked until emphysema has developed and the heart is dilated, satisfactory recovery is much less likely to occur. In most cases in which the lungs are not badly damaged, the chances of recovery, at least before puberty, are very good, provided suitable treatment has been carried out.

Treatment.—The treatment of asthma may be considered under three heads: (1) that of an acute attack which has begun; (2) when an attack threatens; and (3) what is most important, the measures to be taken during the intervals between the attacks, to prevent their recurrence.

(1) *The Acute Attack.*—The patient must be put to bed in an airy room, and an expectorant and diaphoretic mixture given for the catarrh. A large mustard poultice, composed of one part of mustard and from two to four of linseed meal, according to the child's age, often gives great relief. It should be left on until the skin is well reddened, but, of course, must not be repeated too often. Should there be reason to suspect the presence of undigested food in the stomach or bowel, it will be well to administer an emetic or purgative at an early stage.

In a severe case—say, in a child of five or six years—the hypodermic injection of adrenalin solution (3 to 5 minims) or ephedrine ($\frac{1}{4}$ to $\frac{1}{2}$ gr.), is probably the most effectual remedy and

often brings the symptoms rapidly to an end. A hypodermic injection of morphine ($\frac{1}{12}$ gr.), or the inhalation of chloroform, may be similarly useful. In cases in which the symptoms are less urgent, antipyrine (3 to 6 gr.) with chloral (3 to 8 gr.) or bromide of soda (8 to 12 gr.) may be given by the mouth. The fumes of nitre paper or of stramonium leaves, which are constituents of most patent inhalation cures, are often helpful.

(2) *A Threatened Attack*.—In children who are subject to asthma, much may be done to ward off an attack by warmth, prompt attention to the digestion and bowels, and the timely administration of antipyrine, bromide, and iodide.

(3) *Between the Attacks*.—This aims at diminishing their frequency and severity, and, if possible, stopping them altogether.

Older Methods of Treatment.—Formerly, before anything was known about anaphylaxis and its relation to asthma, a number of measures were employed with success in many cases. They are still valuable, and often form the only treatment available.

They may be summed up as follows: (1) Attention to the Hygiene and General Health; (2) Treatment of any Local Disease; (3) Regulation of the Diet and Habits of Eating and Drinking; and (4) Administration of Medicines.

(1) The *Hygiene and General Health* should always receive attention. When possible, it is well to have the child removed to a warm, dry climate; and, in any case, it is important to ascertain, as soon as possible, how far the symptoms are influenced by such climates as are available. In some cases, no change of air has any effect. In others, changes make a great difference in the frequency and severity of the attacks; and removal to a place that suits the child better may stop the asthma altogether. Sometimes the seaside agrees best; often, however, the sea air aggravates the symptoms, and hill air, at a moderate elevation, is more beneficial. Some cases do best in a town, others are only well in the country. When a place is found that suits the child, it is important to let him have the advantage of living there as much as possible, as long intervals of immunity are often obtained in this way, and these go far toward breaking the habit of the disease and preventing permanent emphysema.

(2) Any *Local Disease* that is present must be dealt with. Precautions should be taken to avoid and to treat at once any form of recurrent catarrh. In a few cases operations on adenoids or unhealthy tonsils are followed by improvement—probably because they remove a source of repeated infection. Local suppuration may aggravate the symptoms. If there is indigestion, means should be taken to remove it.

(3) The *Child's Diet and Habits of Eating and Drinking* should be regulated. The meals should be moderate in size, and the intervals sufficiently long to allow the stomach to be emptied before more food is taken. An excess of butcher-meat or of sweets is to be avoided. In older children the food should be given as dry as possible, and fluids mainly when the stomach is empty. Of course, any form of food which has been found to disagree must be strictly forbidden. Parents do not always realise the importance of this obvious indication.

(4) Several *Medicines* are of considerable value. In many cases great improvement follows the regular use of small doses of iodide of potash (1 to 4 gr., twice or thrice daily). Its effect is often striking; even very small doses may enable a child to remain free from the attacks in a place where, without them, they constantly recur. *Liquor arsenicalis* (1 to 3 minims, thrice daily) and cod-liver oil are also beneficial. In some young children with a tendency to "lithæmia," a course of salol (1 to 3 gr.) with bicarbonate of soda (3 to 6 gr.) given thrice daily, an hour before food, will lessen the frequency and severity of the attacks.

The New Methods.—The knowledge of the frequent dependence of asthma on anaphylaxis has led to the introduction of another form of treatment. This has proved of value in many instances, and will probably become increasingly useful when its scope and limitations have been fully realised.

If the skin test shows a decided reaction to the protein of any food which the child has been taking, the effect of stopping it altogether should first be tried. In many cases this is followed by a complete cessation of the attacks. When this occurs, it is desirable, if possible, to attempt to desensitise the patient by accustoming him very gradually to it. This may often be effected by administering it to him in minute doses at short intervals over a long period, until he becomes immunised to it. The initial dose for this purpose must be

very small indeed (*e.g.*, 1 mgrm., or even less, once a day), for it is necessary to begin below the limit of tolerance and work up very gradually to larger doses. The treatment is, of course, very tedious, and requires so much patience and perseverance on the part of those in charge that it is not often possible to get it properly carried out.

When the attacks arise from the inhalation of pollen or of animal emanations, and the patient cannot be kept away from the cause of his attacks, desensitisation may be attempted by gradually increasing hypodermic injections of the protein concerned, at intervals of a few days.

If there is good reason to blame the presence of pathogenic micro-organisms anywhere in the body, the injection of small doses of an autogenous vaccine should be tried, and is often completely successful.

Diseases with Laryngeal Symptoms.

The main characteristic of laryngeal disorders in childhood is their tendency to be associated with spasm; and it is chiefly the various forms of laryngeal spasm that we shall consider here. We shall not take up the important subject of laryngeal diphtheria; and laryngismus has already been dealt with in the chapter on Spasmophilia (p. 541). Simple acute laryngitis, also, which is not uncommon in childhood and which may be met with at all ages—even occasionally in young babies—need not be dealt with, as it does not differ from the same condition in later life. Of chronic laryngitis the same may be said. It may, however, be repeated that when it is met with in early infancy it should always suggest the possible presence of syphilis.

Laryngeal affections, owing to the noise they make, are always likely to be noticed early; and they are apt to cause anxiety, because they suggest the question, "Has the child got croup (*i.e.*, laryngeal diphtheria)?" The most important causes of stridorous breathing in early childhood—apart from diphtheria, laryngismus, and adenoids—are false croup, congenital stridor, enlarged bronchial glands, and laryngeal papilloma.

False Croup (*Spasmodic Croup, Catarrhal Spasm, Laryngitis Stridula*).

This condition may occur any time between the ages of nine months and ten years, or later, but is commonest between two and six. Although it is not really a serious disease, it often gives rise to great anxiety.

Symptoms.—The onset of the attack is sudden and almost always occurs at night. The child, who has usually shown signs of slight catarrh, wakens flushed, frightened, and with a metallic cough and loud croupy breathing. The cry is not much affected, but the child is somewhat feverish (about 101° F. or so), and perspiring, and seems distressed. The fauces show only a slight catarrh.

After an hour or two he becomes quieter and falls asleep. When he does so, or even when he merely becomes less anxious, the respiration is at once much easier—showing distinctly the nervous character of the ailment. A second attack often follows on the same night, and the symptoms are also apt to recur about bedtime on the following evening. Thereafter they generally give place to those of an ordinary cold. A child who has had one attack of this kind is liable to have others. Occasionally the disease recurs at intervals for years; and often more than one child in a family is affected.

Diagnosis.—False croup is to be distinguished from diphtheritic laryngitis by its sudden commencement, its less severe character, and the more markedly spasmodic or intermittent nature of the symptoms; also by the appearance of the fauces. In doubtful cases the history of previous attacks of a similar nature in the patient, or even in other children of the family, is reassuring. It must be remembered, however, that in rare cases diphtheria may begin with symptoms very like those of false croup, and that it is comparatively common to have attacks of *measles* and *whooping-cough* commencing in a similar way.

Laryngismus should never be mistaken for this condition. The patients are younger, the spasms are shorter in duration, though recurring frequently through the day. Cough, fever, and coryza are absent, and other manifestations of spasmophilia *e.g.*, the facial phenomenon, are usually to be found.

Treatment.—The main treatment of false croup consists

in the application of warmth, internally and externally, to encourage the secretions and to soothe the child. Warm drinks are useful, and also the application of hot fomentations or poultices to the neck. It is also usually wise to put the child into a warm bath, and in severe cases, to erect a steam tent. A simple expectorant mixture may be given. Should there be reason to suspect the presence of undigested matters in the stomach or bowel, an emetic (pulv. ipecac. 5 gr.) is indicated or a dose of castor oil. Neither of them should, however, be given as a mere matter of routine.

Congenital Stridor (*Infantile Respiratory Spasm*).

This comparatively harmless affection is characterised by crowing breathing, a peculiar formation of the vestibule of the larynx, and, in advanced cases, by some thoracic deformity. The characteristic stridor commences at birth, or, at latest, within a week or two after the child is born.

Symptoms.—The stridor consists in a croaking sound which accompanies inspiration and rises to a high-pitched crow on quicker or deeper breathing. Expiration is usually noiseless, but sometimes, when the inspiratory noise is loud, it is accompanied by a short croak, and there is considerable inspiratory indrawing of the sides of the chest. There is, however, little movement of the alæ nasi and no cyanosis. The child is evidently in no distress, and looks about him quite unconcerned. The loudness and pitch of the crowing vary from time to time, and even in severe cases there are short periods of intermission during which no sound is heard.

In slight cases the noise ceases during sleep, but when the stridor is severe it may last all the time. Any mental excitement increases it. It is not diminished by closing the nostrils or the mouth, but the introduction of an intubation tube stops it at once (Variot). The cry is loud and clear, and if a cough is present it is quite normal in character.

Ashby has described a difficulty in swallowing as occurring in a few of these cases¹; and Dan McKenzie is of opinion that this is not uncommon.²

During the early weeks of life the stridor increases in

¹ *Brit. Med. Journ.*, Nov. 1906, 1485.

² *Journ. Laryng. and Otol.*, 1925, xl., No. 5.

loudness with the child's growth in strength. Thereafter it generally remains about the same till some time after the sixth month, when it spontaneously diminishes; and it passes off before the end of the second year in most cases. Latterly it is only heard on occasions of special exertion or excitement.

In all cases which have lasted for any time there is a characteristic deformity of the upper aperture of the larynx (Fig. 209), which consists essentially in a great exaggeration of the normal infantile peculiarities of the part (p. 606). The epiglottis is folded on itself, so that the aryteno-epiglottic folds

lie close together and render the transverse diameter of the upper aperture of the larynx extremely narrow. In most cases there are no adenoid growths and the fauces are normal. If the stridor is severe and lasts for any time, a distinct degree of pigeon-breast is produced.

As the child grows up, the vestibule of the larynx retains the characteristic deformity long after the stridor has ceased. It can often be recognised in children of nine or ten, and probably much later. When the breathing becomes normal, the thoracic deformity steadily lessens and may finally disappear.



FIG. 209.—Larynx of baby of 3 months who had suffered from Stridor from birth, and died of exhaustion following diarrhoea.

Causation.—The etiology of this condition has been much discussed, but it is not yet thoroughly understood. The stridor has been attributed to posticus paralysis (Robertson) and to adductor spasm from adenoid growths or other local irritation (E. Smith, Löri), to enlargement of the thymus (Avellis, Hochsinger), and to congenital malformation of the upper aperture of the larynx (Lees, Sutherland and Lack, Variot, Refslund). John Thomson and Logan Turner¹ have endeavoured to show that, while the deformity of the larynx and the soft

¹ Thomson and Turner, "On the Causation of the Congenital Stridor of Infants," *Brit. Med. Journ.*, 1st Dec. 1900, ii., 1561; Thomson, *Edin. Med. Journ.*, Sept. 1892, xxxvii., 205; *idem*, Art. "Stridor des Nouveau-nés," *Grancher and Comby's Traité des Maladies de l'Enfance*, 2^{me} ed., iii., 113; Turner, *Brit. Med. Journ.*, 24th Nov. 1906, ii., 1485.

collapsible character of its structures are not without importance in the etiology of the stridor, the part which they play is only a secondary one; and that the essential and primary factor in the disease is not structural but functional. They hold that the primary cause of the obstruction is an ill-co-ordinated spasmodic action of the muscles of respiration, a choreiform respiratory spasm analogous to stammering — as distinguished from a laryngeal spasm. That such jerky forcible respiration would



FIG. 210.—Larynx of new-born child; showing normal conformation.



FIG. 211.—The same; showing effect of sudden suction through metal tube on the shape of its upper aperture.

be likely to cause exactly this deformity is proved by a simple experiment. The larynx of a new-born child is removed along with the neighbouring parts (Fig. 210). A bent metal tube, 18 in. in length, is then introduced into the lower end of the trachea; and in order to keep the chink of the glottis open a small piece of rubber tubing is at the same time placed between the vocal cords. When forcible inspirations are made through the tube, it is found that with each a striking alteration occurs in the upper aperture of the larynx, and that in many cases (as in Fig. 211) the resulting deformity is indistinguishable from that seen in cases of congenital stridor.

Diagnosis.—The recognition of congenital stridor is easy

in typical and uncomplicated cases. Taking into account the character of the stridor, the age at which it began, the normal sound of the cry, and the absence of distress and of any other sign of disease, one can readily exclude such conditions as laryngismus, laryngitis, and laryngeal papilloma.

Conditions which may give rise to somewhat similar symptoms are hyperplasia of the thymus, enlargement of the bronchial and tracheal lymphatic glands and adenoid growths, all of which occasionally occur in young babies.

The differential diagnosis between thymic stridor due to an enlarged thymus and congenital laryngeal stridor has already been discussed (p. 497).

The cases of noisy respiration due to *enlarged bronchial and tracheal glands* usually differ from those of congenital stridor in the following points: (1) The stridor is lower in pitch; (2) it is generally louder during expiration; (3) the up-and-down movements of the larynx accompanying the noisy breathing are less extensive; (4) the voice and cry are generally affected; and (5) there is usually some cachexia present from the cause of the enlarged glands.

The croaking in cases of *adenoid growths* in young babies sometimes resembles that of congenital stridor rather closely. In these cases also, however, it has a distinctly lower pitch, and there is usually a hoarse cry. Mouth-breathing and other signs of nasal obstruction are also generally, though not always, noticeable. The form of croaking, unlike that of the real congenital stridor, is cured or greatly relieved by operation on the adenoids.

Prognosis.—Congenital stridor, when uncomplicated, is always recovered from. It must, however, be remembered that if babies with this condition acquire any acute respiratory disease, the abnormal state of the larynx may act as a serious complication.

Treatment.—The only rational treatment of simple cases of congenital stridor consists in careful regulation of the diet and of the general hygiene.

Laryngeal Papilloma.

This is a rare affection, and does not generally produce symptoms till the child is two or three years old or more, although the disease is believed to originate at an earlier

period. The main symptoms which characterise it resemble those of severe chronic laryngitis—stridorous breathing, a hoarse cry, and a croupy cough; and they gradually get worse in spite of treatment. In young children it may be practically impossible at first to make sure of the diagnosis.

Treatment.—The surgical removal of the growths is usually now carried out through the mouth, by the direct method. If they recur, the operation may have to be repeated. Treatment by radium has recently been found satisfactory in some cases.

CHAPTER XXVII

SYMPTOMS AND SIGNS CONNECTED WITH THE NERVOUS SYSTEM

THE majority of alarming nervous and mental symptoms in young children are not caused by organic disease of the nervous system at all, but arise from disorders of other systems, or from such general morbid conditions as pyrexia, toxæmia, or uræmia. It takes very little to disturb a child's nervous system so thoroughly that the localising symptoms of disease in the chest, abdomen, or elsewhere become obscured. We have, therefore, to get into the habit of discounting the nervous manifestations present before we can justly estimate the meaning of the symptoms of acute disease in little children.

Structural disease of the nervous system is, however, not uncommon in early life, and occasionally gives rise to very obvious symptoms; but often its clinical manifestations are curiously slight and equivocal.

Examination of Nervous Cases.

When a child has to be examined as to the presence of disease of the nervous system, the necessary inquiries may be divided into three groups:—

1. Investigation as to the occurrence of *cerebral* or *nervous symptoms*. We may ask about any change noticed in the child's disposition and temper and whether he has been drowsy or delirious. We should also inquire about pain in the head or elsewhere, photophobia, giddiness, and sudden screaming. He may have been heard to sigh, or grind his teeth, and he may vomit, be constipated for the first time, or have convulsions.

2. The patient has to be examined for *physical signs*. We have already discussed the significance of the characteristic change in the physiognomy, of distended cranial veins and a bulging fontanelle, and of a slow and irregular pulse.

The state of the pupils must be noted and the presence

of any nystagmus or squint; and the ophthalmoscope should invariably be used. A distinct degree of cervical rigidity or opisthotonos is always of great importance, although this may be present in an exquisite degree in such diseases as pneumonia and pyelonephritis.

The limbs are to be examined as to their sensibility and the presence of paralysis, rigidity, ataxia, and tremor or other involuntary movements. The state of the superficial reflexes and knee-jerks should be ascertained and the electrical reactions tested.

3. Previous occurrence of *such illnesses as are known to be often followed by nervous or mental disease* should be inquired about. Thus, the former presence of chronic middle-ear disease has great importance in suspected cerebral abscess, and if tuberculous meningitis is possibly present, the fact of the symptoms following whooping-cough or measles is in favour of this diagnosis.

Some of the more important symptoms and signs of nervous disease may now be considered more fully.

Changes in Disposition and Temper.

Great irritability with a marked change in character is often noticed during the prodromal stage of tuberculous meningitis. This is not always, however, of much help in diagnosis, as somewhat similar mental disturbances are equally characteristic of various other abnormal conditions. Unwonted irritability is seen during the onset of most acute ailments and in a more chronic form in some cases of dyspepsia, chronic diarrhoea, chronic nephritis, and diabetes mellitus.

Screaming.

In young infants, continually recurring paroxysms of screaming may be caused in various ways. Most frequently they are due to flatulence and colic (p. 149); occasionally they are set up by the irritation of uric acid in the urine or of preputial irritation; and sometimes they are a symptom of an ill-developed brain with severe mental defect.

In older infants and young children also, screaming is a common and characteristic symptom. Its causation differs greatly in different cases.

A. It may be due altogether to *bodily causes*.

1. It is often a natural manifestation of acute spontaneous pain in some part of the body—such as is produced by intestinal or renal colic, rectal spasm, acute otitis media, or any other distressing form of spasm or acute inflammation.

2. It may occasionally arise from intracranial disease without any pain. A shrill piercing scream—the “hydrocephalic cry”—is one of the classical symptoms of tuberculous meningitis; and a similar loud cry sometimes occurs in chronic hydrocephalus when the fluid is increasing rapidly.

3. When violent outbreaks of passionate screaming occur in little children without apparent cause—especially in those who are well brought up and have not hitherto had any similar attacks—it is most important to remember that, in a large proportion of cases, some latent disturbance of the general health is to blame for such attacks, and that simple medical treatment will usually stop their recurrence. Generally, the fault is connected with some form of indigestion, but it may be due to toxæmia, such as arises from *Bacillus coli* invasion of the urinary tract, or staphylococcus infection of the throat or nose. Such ailments may set up a continuous, though probably unrecognised, feeling of discomfort with great hypersensitiveness, so that the most trivial annoyances arouse violent emotion. In dyspeptic cases, the common nursery prescription of a dose of castor oil often does good for the time; and in those due to acidosis, the regular use of bicarbonate of soda and salol will usually work wonders in restoring the child's equanimity and preventing any recurrence of the attacks. When the irritability is due to a bacterial infection, minute doses of a suitable vaccine may be equally effective.

This type of screaming is common in mentally defective children; but, in them especially, it is often impossible, before the effect of treatment has been tried, to make out whether the main cause of the disturbance is bodily or mental.

The paroxysms of screaming which are associated with night terrors may also be included in this group.

B. The screaming may be due altogether to *mental causes*.

Recurrent paroxysms of screaming are in many nurseries a familiar climax to states of ill-temper with loss of self-control, and they are usually due largely to want of sense and sympathy on the part of those in charge. In some children, such

“tantrums” are successfully treated by prompt corporal punishment. In many instances, however, in thoughtful children who are not spoiled, the outbreak is not due to naughtiness: the child himself is puzzled by the vagaries of his subconscious self and seems as much troubled by his own behaviour as his parents. In these circumstances corporal punishment is both unsuitable and useless; and the management of the attacks requires much knowledge of the individual child and the highest type of mothercraft. It is necessary to bear in mind the child’s instinctive tendency to self-display, and the gratification he derives from making an impression on his seniors. This element may play only a small part in some children, in others it bulks largely. In all cases it is advisable that any worry or annoyance that is felt should be concealed, and that the screaming attacks should never be spoken about in the child’s hearing. Often the mother learns to recognise premonitory symptoms which indicate that an attack threatens, and she may be able to avert it by getting the child to do at once some little action which will divert his mind from the subject. She may, for example, get him to take a definite number of long breaths, to go and sponge his face with cold water, or to fetch something from the garden.

In some instances the presence of morbid fears may be the real cause of the screaming (p. 696).

Drowsiness.

Drowsiness is an important symptom in intracranial disease. In tuberculous meningitis it sometimes occurs at the onset, and it is always a prominent feature in the later stages. After an epileptiform convulsion, the child is often drowsy for many hours; and, if fits occur in a long series at short intervals, the drowsiness may be so severe and last so long that, even when the brain is not permanently damaged, the child may look as if he were becoming an idiot (Fig. 212).

Extreme drowsiness, often passing into stupor, is a characteristic phenomenon in thrombosis of the cerebral sinuses and in encephalitis lethargica.

In very many cases, however, drowsiness is not a sign of brain disease at all. It is characteristic of various food disorders, acid intoxication, and uræmia, and occurs in hepatic and

gastric maladies, and to some extent in most cases of high temperature.

It may also, of course, be the result of medicine (*e.g.* bromide); and, when it is present in severe illness, it is always well to make sure that it is not merely due to excessive doses of the panacea for all ills, *viz.*—alcohol.

Coma.

Coma is almost always a symptom of the gravest significance. It occurs in diabetes, in uræmia, in liver atrophy, in intracranial injuries, and many other diseases, including eclampsia, and in various forms of poisoning.



FIG. 212.—Prolonged Stupor following repeated Idiopathic Convulsions in a boy of 6 months. The child entirely recovered.

Delirium.

Delirium is not uncommon in feverish conditions in childhood, and it may be recognised even in young babies by watching their gestures and expression (Fig. 11, p. 11). Its occurrence in any case depends less on the degree of fever than on the kind of nervous system the child has, and on the character of his illness.

Some children become delirious if their temperature rises at night to 101° or even to 100° F., while others can stand a temperature of 106° F. without becoming light-headed. Proneness to delirium at comparatively low temperature is probably an indication of an unstable nervous system.¹ Although a recognised symptom in both tuberculous and non-tuberculous meningitis, delirium is much more frequently an indication of general toxæmia than of intracranial disease. It is common, for example, in peritonitis,

¹ Clouston, *The Neuroses of Development*, Edin., 1891, 12.

in liver atrophy, and in all sorts of septic infection, in enteric, in erysipelas, and in severe cases of infective disease of any kind. It is probably most common in pneumonia. It is a curious feature of some febrile diseases such as pneumonia, ileo-colitis and pyelonephritis that delirium is most prone to occur at the commencement of the illness. In pneumonia the delirium may only appear after the crisis.

The possibility of delirium being caused by atropine or belladonna is not to be forgotten.

Pain.

When *persistent local pain* is complained of by young children, it almost invariably signifies the presence of organic disease; and, if the cause is not obvious, the child must be kept under observation. Pain of this kind must never be regarded as a trivial matter. On the other hand, pain is often absent in young children in diseases in which we should expect to find it in adults, such as pleurisy, herpes zoster, and otitis media. We must also remember that young children often say they have pain, when little or none is present, because they have been accustomed to hear their mothers do so; and that they not infrequently deny having pain when they are really suffering, because they do not clearly understand the meaning of what is asked. It is therefore best to found our diagnosis of this symptom more on what we observe of the patient's expression and behaviour than upon his complaints.

A distressed expression, with uneasy working of the mouth and lifting the hand to the jaws or ears, is seen in teething babies, and also in those with acute otitis.

The paroxysms of frightened crying, with flexing of the lower limbs and arms, clenching of the fists and the subsequent passage of flatus, which indicate intestinal colic, have been already alluded to (p. 150). These are readily distinguished from the attacks of screaming and stiffening of the back, with the lower extremities fully extended and pressed together, which are seen in rectal spasm (p. 137). In renal colic also the lower limbs are generally not flexed, and there is often tenderness in one or other loin, or over the distribution of the twelfth dorsal and first lumbar nerves above the groin;

the testicle is also tender and retracted, and the breathing quickened.

The characters of acute pleuritic pain have already been described (p. 7). It causes an expression of great distress and anxiety because the patient has to restrain his inclination to cry, on account of the pain which every full breath causes. This, with the dilated and working *alæ nasi* and the rapid and shallow breathing, is quite characteristic. Pain due to disease in one or other *pleura* is sometimes referred by the child to the middle-line in front, often to the epigastrium. Occasionally it is felt quite low down on the side of the abdomen, and when on the right side may be mistaken for that of appendicitis. Another exceedingly distressing and sickening form of pain is the kind of angina which occurs in some cases of congenital heart disease (p. 584).

Abdominal pain is often, of course, due to digestive disturbance, and it is also frequently caused by spinal caries. Pericarditis, it should be remembered, is often accompanied by pain referred to the abdomen. Pain in the *thigh* or on the *inner side of the knee* is an early symptom of hip-joint disease; but, in this situation or lower down (even in the foot), it may be due to spinal caries, and it sometimes occurs at a stage when no local abnormality of the spine can be discovered without an X-ray examination.

The shock which severe pain inflicts on a child is so great that it should never be allowed to continue long without relief; therefore prolonged and painful surgical dressings should usually be done under chloroform.

Headache.

In school children headaches are often complained of, and when they are recurrent they leave distinct traces on the *physiognomy*. Dr Francis Warner says: "It is not uncommon to observe that a child looks as if he had a headache. Analysing such faces you may soon observe a look of depression, heaviness, and fullness about the eyes, especially about the lower eyelids: this sign is usually bilateral, and is due to a relaxed condition of the muscle (*orbicularis*) which surrounds the eyelids. If the patient can be made to laugh, the muscle becomes energised, and the expression of headache is lost for the moment. This sign is often best seen in the profile view."¹

¹ *The Children: How to Study Them*, London, 1885, 51.

In all cases of headache the teeth should be examined for caries. Severe headaches occur in uræmia, malaria, anæmia, and plumbism.

One of the commonest forms of headache is that due to *refractive anomalies*, especially slight degrees of hypermetropia and astigmatism. In this case the headache is usually present on school-days and absent during the week-end and holidays. It is rapidly cured in most cases by the use of suitable spectacles.

The presence of *adenoid growths* also gives rise to some headache in a large proportion of cases (50 per cent.—Crowley).

Dyspepsia is another common source of headaches. In many cases, whether there is a discoverable local cause or not, the patients are delicate children who are obviously being *overworked at school* and having too little fresh air and exercise.

Migraine sometimes begins in young children as a recurrent acidosis and is often accompanied by a rise of temperature. As the child grows older, however, the attacks assume the ordinary afebrile character seen in adults (p. 559).

Recurrent headaches in children should be treated with soda (p. 563) during the intervals between the attacks. They are sometimes benefited by a combination of liquid extract of ergot (10 to 20 minims) and solution of strychnine (2 to 3 minims) (E. Smith).

In infants, headache may be the result of coryza or pyrexia, or of some gastric or hepatic disorder. If severe or persistent—especially if accompanied by vomiting—it is often a sign of meningitis or intracranial tumour.

Giddiness.

Vertigo is sometimes found in young children. When in bed or sitting on his mother's knee, the child cries out that he is falling, or that the roof or furniture is moving. Giddiness may also be a symptom of *tuberculous* or of *posterior basic meningitis*. It occurs sometimes during the onset of various infectious diseases, such as mumps, influenza, and measles. We have also known it occur, off and on, for months, in young children with obstinate constipation from *disordered digestion*, and nothing worse. When vertigo is present, the condition of the *ears* should always, of course, be investigated.

Infantile Convulsions.

It is hardly necessary to give a detailed account of a well-marked infantile convulsion, as its phenomena are just those of an ordinary epileptic seizure. In the worst forms the tonic and clonic movements may be very severe, and the succeeding loss of consciousness profound and long continued. In other cases there may be just the slightest momentary unconsciousness and no visible jerking at all. Slight and severe attacks often occur in the same child at different times.

Causation.—Like all nervous disorders which consist in disturbance of function, convulsions have often a multiple causation. That is to say, in order to explain their onset we must take into account not only one or more exciting causes, but also a number of predisposing conditions. This point is of some practical importance, because we may often succeed in stopping the recurrence of the convulsions by curing one or more of the causes, even although the others are beyond our treatment. The division into predisposing and exciting causes is convenient, although it must be admitted that it is not always easy to draw the line clearly between the two.

Of *predisposing causes* four may be mentioned: (1) There is, of course, the *age* of the patient—the state of development of the infant's nervous system predisposing him to all kinds of convulsive attacks. (2) Certain general diseases may predispose—especially *spasmophilia* (p. 538). This is the most important of the predisposing causes in young children and also that which is most amenable to immediate treatment. The tendency to convulsions in this condition almost always disappears rapidly under suitable treatment, even although obvious sources of peripheral irritation persist. (3) A very important predisposing element is an *inherited nervousness* of constitution. Some children are hereditarily so nervous that even a slight rise of temperature or peripheral irritation will bring on a fit. This state of nervousness may be found in children who seem otherwise strong, and sometimes many members of a family have it. (4) Another predisposing condition is a permanently *damaged state of the brain* from any cause—quite apart from any recent changes. An area of cortical sclerosis, for example, even when it does not seriously affect the mental functions, is often accompanied by a tendency to convulsions. In the same

way, nearly all the developmental and other lesions which produce imbecility predispose to convulsions on slight provocation. Their recurrence in young children should, therefore, always lead to a careful consideration of the child's mental state.

Possible *exciting causes* are very numerous. The most important of them may be classed in one or other of three groups: (1) *Intracranial causes* (diseases, injuries, or circulatory changes).—Such, for example, are concussion, hæmorrhage, tumour, abscess, meningitis, and encephalitis; also the cerebral congestion which occurs in whooping-cough and in some cases of congenital heart disease, and the cerebral anæmia which accompanies severe diarrhœa and loss of blood. (2) *General acute morbid conditions*.—A sudden rise of temperature, such as would produce a rigor in an adult, will often cause a convulsion in a child. This is seen in pneumonia and in certain of the exanthemata, especially scarlet fever. In uræmia, fits are not uncommon, and a large number of poisons, both metallic and vegetable, may cause them. (3) *Peripheral nervous irritation*—often of obscure origin. Undigested matters in the bowel or stomach, painful lesions in connection with the teeth, the ears, or the skin readily start convulsions in spasmophilic and neurotic infants.

Diagnosis.—There cannot usually be much difficulty in recognising a convulsion, if you see it. When, however, you have only the mother's description to go by, it may be impossible to be quite sure as to the nature of the "fit." Attacks of *petit mal* often pass unrecognised for months from the idea that they are merely slight faints. On the other hand, spasms of colic and laryngismus, and also rigors, are sometimes mistaken for fits. On several occasions we have seen cases of masturbation in female infants, who were being carefully nursed and having bromides and sympathy administered under the impression that they were suffering from a distressing variety of infantile convulsions.

Diagnosis of the Cause.—Whenever the urgency of the symptoms has abated, the cause of the attack must be investigated.

1. There is, to begin with, the patient's *age*. Should the attack come on within the first fortnight of life, the likelihood of its being due to a birth injury is to be remembered, though convulsions from dyspepsia and other causes, quite apart from

trauma, often begin soon after birth. To this subject we shall return later. Those convulsions which begin after the second week are much less likely to have anything to do with a birth injury. They may arise from a defective brain, from dyspepsia, and from various other causes. The great majority of fits seen in normally developed babies between six months and two years belong to the spasmophilic class.

2. The *character of the fit* often gives us little help in the diagnosis, though occasionally it does shed some light on the question. If, for example, the features of the attack are those of *petit mal*, this generally, although not always, indicates a serious cerebral defect. Inherited lead poisoning sometimes causes attacks in which respiratory symptoms predominate. If the fit is Jacksonian in character, this suggests a cortical lesion. If it is followed by prolonged unconsciousness, an organic cerebral cause becomes probable although not certain. Unilateral or asymmetrical movements do not necessarily, in young children, indicate a unilateral organic cause.

3. The presence of *symptoms of any bodily disease* that is known sometimes to cause fits is, of course, of great importance. Thus, fever with rapid respiration or a sore throat may point to pneumonia or scarlet fever. A bulging fontanelle, head retraction, or paralysis, with a history of previous vomiting and headache, would suggest meningitis; while abdominal distension and malnutrition, along with other dyspeptic symptoms, would naturally point to there being some connection with the alimentary canal.

Prognosis.—When mental defect is present after a series of convulsions, it is generally probable that it existed before, and that it should be looked upon rather as the cause than as a result of the fits. Certainly, however, in some cases the intense cerebral congestion and excitement accompanying the seizure seriously injure the brain tissue, either by causing hæmorrhage or otherwise. The recurrence of convulsions is accordingly often followed by steadily increasing dementia, even in cases in which no naked-eye change is afterwards discoverable. Occasionally, although rarely, the attack may be fatal. This probably occurs most frequently in cases of spasmophilia in which the fits are complicated by laryngismus. These considerations emphasise the importance of using active measures to stop the attacks as soon as possible. In many cases, also, temporary

damage is done, and one or other cerebral function may remain in abeyance for some weeks, or months, after a severe convulsive attack. Thus, passing hemiplegia, or aphasia, or blindness may be due to temporary exhaustion produced in certain areas by the nerve storm. Similarly, a condition of extreme intellectual dullness occasionally results, which is entirely recovered from, even though it has lasted for weeks.

Treatment of the Attack.—If the convulsion lasts long enough to allow time for treatment, it is generally advisable to begin by putting the child into a mustard pack or hot bath. It will probably be good for him, and it will certainly soothe and relieve his alarmed relatives, who generally need something to do to take up their attention.

If the convulsive movements continue for more than a few minutes, or if they go on recurring at short intervals, more active sedative measures are called for. Chloroform inhalation is one of the best of these, and it is quite safe and often successful. Chloral hydrate is also very useful, and its influence lasts longer than that of chloroform. If the child cannot swallow, it may be given hypodermically (2 or 3 gr.) or introduced into the bowel through a rubber catheter. For rectal injection, 5 gr. is the dose for a baby of six months, and 10 gr. for one of a year old. In severe cases the most effectual treatment of all is the hypodermic injection of morphine. Of this, $\frac{1}{24}$ gr. may be given to a well-grown baby of a year, and the dose may be repeated in half an hour if no effect is produced. Morphine should not be given to weakly or undergrown babies.

The treatment of convulsions, however, is less important than the prevention of their recurrence. As this depends mainly on the type of case present, we shall consider briefly a few more or less distinct types that may be met with.

Various Types of Convulsion Cases.

1. **From Birth Injury.**—If convulsions appear in a new-born baby within the first week or so of life, it is natural to suspect that they may be due to intracranial hæmorrhage or other birth injury. This suspicion is greatly strengthened if the labour was premature or difficult, and especially if the child had fits or difficulty in breathing after birth (p. 727).

If on examination of the cerebro-spinal fluid it is found blood-stained or xanthochromic, injury of the brain can be diagnosed with some probability.

The *treatment* should be generally purely expectant. The infant is to be kept quiet and warm, and everything possible done to favour his nutrition. Sedatives are not usually called for. Cushing has recommended¹ opening the cranium in these cases. While this suggestion is interesting, its advisability in most cases cannot be said, as yet, to have been established, especially when the diagnosis cannot be made with absolute certainty.

The *prognosis* must also be guarded. It is probable that many of the children who have traumatic cerebral hæmorrhages at birth get quite well; but certainly many of those who recover show symptoms of paralysis, epilepsy, or mental defect in later childhood.

2. **From Dyspepsia.**—In many infants who have convulsions the attacks are clearly due to dyspepsia. Whether they arise reflexly from local irritation in the bowel, or are caused by auto-intoxication, need not be discussed here. In these cases the fits may begin soon after birth, or indeed at any time during infancy.

The main *treatment* consists in thorough regulation of the diet, along with the judicious use of calomel, antacids, and stomach-washing; and, when possible, a wet nurse should be obtained. Generally, no sedatives are required; but a few doses of chloral may be useful at first.

The *prognosis* depends on the progress of the dyspepsia. There is no likelihood that the brain will be seriously or permanently damaged.

3. **Idiopathic Convulsions.**—The next group of cases may, in the meantime, be conveniently called “idiopathic convulsions,” because no central, peripheral or other cause has as yet been proved to account for their occurrence.

The patients are mostly boys, either breast- or bottle-fed, and they have shown no previous sign of disease. The attacks, which may be unilateral, often begin as early as the first week and generally within the first few months. The convulsions are slight in character and of short duration; and at first only two or three may occur in the day. They usually, however,

¹ *Amer. Journ. of Med. Sci.*, 1905, cxxx., 563.

increase rapidly in number till there are as many as twenty or even forty in the day ; and, if the case is not efficiently treated, this number may continue for weeks or months.

Bromides have little or no effect, and to try alterations of diet when none are indicated is merely to lose time. It is *very important* to get the infant thoroughly under the influence of chloral as soon as possible ; and it can easily be given by the mouth. In the youngest babies, 1 gr. every two hours, and in children of one or two months, $1\frac{1}{2}$ or even 2 gr. is not too large a dose. These doses should be continued until the fits have ceased for at least twenty-four or thirty-six hours, and then gradually diminished in frequency. If the first dose is not enough, the amount must be cautiously increased until the baby is *almost* too drowsy to swallow. The greatest care must, however, be given to the feeding when the child becomes drowsy, as otherwise there is a considerable risk of an inhalation pneumonia being set up. Usually, after the chloral has been given for three or four days at most, the fits cease to return when it is stopped, and the child remains quite well. If the chloral is not continued long enough the fits return.

The usual completeness and permanence of the recovery of these cases after a short course of chloral are very striking. The cause of the attacks is obscure, but they are probably best explained by the hypothesis that the drug prevents, for the time being, the toxic action of some absorbed protein on the nervous system, and so gives the child's tissues a chance of becoming immunised to it.

The *prognosis* is generally favourable. A large majority of the cases recover completely, and, although some of the babies may remain drowsy and stupid for weeks after the fits have ceased (Fig. 212, p. 664), it is very rare for any permanent injury to be done to the brain. It must, however, be guarded, partly because babies at this age are so feeble that intercurrent affections are to be feared, and partly because there is reason to believe that if the fits are allowed to continue for a very long time unchecked, the brain cells may possibly undergo atrophy and idiocy result. Another reason for a guarded prognosis is the difficulty of being quite sure of the diagnosis. We have seen two cases in which convulsions, apparently of this nature, ushered in acute miliary tuberculosis in infants of four and a half and six weeks respectively.

4. **From Spasmophilia.**—The commonest kind of convulsions met with in a city practice in this country are those which occur about the age of teething, as a symptom of spasmophilia (see Chap. XXII., p. 538).

5. **From Congenital Cerebral Defect.**—In many cases the occurrence of convulsions in an infant is the first, and it may be, as yet, the only sign of idiocy. Generally, the seizures, in these circumstances, take the form of *petit mal* to begin with. The baby is seen from time to time to give a sudden jerk forwards of his head and shoulders (ventral or “salaam” fits). These contractions may recur several times in succession. After this, he may be unconscious for a minute or two with heavy breathing, and he often cries bitterly. As he grows older his “turns” often become more obviously epileptiform in character, and develop into ordinary convulsions; or they may cease.

The occurrence of such attacks is a bad omen for the child’s future. They tell of a serious defect of the brain, and their frequent recurrence is always followed by further mental deterioration. The character of the fits, and the usual signs of mental backwardness, soon render the diagnosis easy.

The results of *treatment* are most disappointing. Sedatives have only a slight and temporary effect. If they are pushed, they upset the digestion and do more harm than good. Frequent changes to the country, and at times the administration of iron or some other tonic, form generally the best treatment. In a very few cases the administration of thyroid does good for a time.

6. **From Congenital Syphilis.**—Another variety of fits, indicating severe structural disease of the brain, is sometimes met with in syphilitic babies of a few months old.¹ In these cases the convulsive movements, which are often very slight, are generally one-sided to begin with, or there may be only a series of twitchings of one arm or leg. They may be accompanied by a momentary loss of consciousness or only by a dazed look. Later, twitchings appear on the other side of the body, and the limbs first affected become flexed and contracted. The child’s intelligence generally deteriorates.

The cortical lesion which gives rise to these symptoms consists in a patchy softening of the grey matter with

¹ Ashby, “On Convulsions during Infancy and Childhood,” *Lancet*, 21st Jan. 1905, 136.

degeneration of the arteries, and it ends in sclerosis. The resulting dementia is severe in degree and permanent. Treatment seems to have little effect.

7. **From Inherited Lead Poisoning.**—In one case observed by one of us (J. T.),¹ a painter who suffered severely for years from unrecognised lead poisoning and ultimately died of it, and whose wife had remained quite healthy, had four consecutive babies who died of fits so peculiar in character that they seem worth mentioning here.

The infants, until shortly before death, seemed well in all other ways, and were unusually intelligent. The fits began between the third week and the sixth month. They set in usually during sleep with sudden restlessness, dyspnoea, and stertor, but without any laryngeal spasm. The child then gave a loud scream, stopped breathing, stiffened, and lost consciousness. There were no clonic movements. After a minute or so the respiration began again and became quick and easy, and the baby wakened, took the breast, and fell asleep.

The attacks had the same characters in all the four children, though they varied in frequency and in severity at different times. In spite of treatment they became longer in duration and more severe; and the children all died in one of them. The ages at death varied from eight to eighteen months.

State of the Pupils.

The size of the child's pupils and the range of their reaction to light are relatively small during the early weeks of life, but they increase steadily after the first month.²

Contracted pupils are seen normally in sleep; they are met with during waking in the early stage of meningitis, in opium narcosis, and in other conditions.

Widely-dilated pupils reacting little or not at all to the action of light, occur in the later stages of most serious cerebral diseases, and also in "hydrocephaloid." Inequality of the pupils is observed in serious cerebral disease and also in cases of affection of the sympathetic in the neck.

¹ J. Thomson, *Brit. Journ. Child. Dis.*, Oct. to Dec. 1923, xx., 193.

² H. Pfister, "Ueber das Verhalten der Pupille und einiger Reflexe am Auge im Säuglings- und frühen Kindesalter," *Archiv. für Kinderheilk.*, 1899, xxvi., 11.

Rhythmical contraction and dilatation of the pupils (hippus) is observed occasionally in cases of spasmus nutans.

Absence of the light reflex with persistence of that on accommodation (Argyll Robertson pupil) is significant of syphilis, and absence of both light and convergence reflexes of disease of the optic nerve.

Strabismus.

The term "squint" is commonly applied to any deviation observed in a child's eyes. Such deviations are met with as the result of three distinct conditions:—



FIG. 213.—Tumour of Medulla, involving nuclei of 6th and 7th nerves. (Girl of 2 years.)¹

1. *Simple disturbance of co-ordination* of the ocular movements. This is not real strabismus. It is often normally present in young babies, especially during high fever, quite apart from head mischief, as well as in cases of meningitis and other intracranial disease.

2. *Ordinary convergent strabismus* is commonly due to hypermetropia. It may begin after a convulsion or during an attack of spasmus nutans, and it is often present in cases of cerebral injury at birth.

3. *Paralytic strabismus* is most frequently seen as a symptom of tuberculous meningitis or of cerebral tumour (Fig. 213).¹ Occasionally it is caused by diphtheritic paralysis, in which case it is generally slight in degree and reveals itself mainly by the diplopia which it occasions. A temporary diplopia is characteristic of the early stage of encephalitis lethargica. We have seen transitory paralysis of the external rectus under other conditions, *e.g.*, once in the course of croupous pneumonia and once during an obscure feverish attack which was possibly influenzal.

¹ J. Thomson, *Trans. Med. Chir. Soc., Edin.*, 1891, x., 210.

Nystagmus.

We may divide oscillating movements of the eyeballs into "nystagmoid movements" and "nystagmus proper." The former term may be used to describe such aimless shaking of the eyes as is seen in some idiots, and the rhythmical twitching which occurs when a patient with conjugate deviation tries to look straight forward and his eyes automatically jerk back to their former position.

Nystagmus proper in childhood includes two conditions—"ordinary nystagmus" and "the nystagmus of spasmus nutans." These differ widely from one another in causation, significance, and prognosis.

Ordinary nystagmus either dates from the earliest infancy or is acquired later. In the former case it is generally due to some local condition which has interfered with the infant's sight at the time when he should have been learning, by the aid of vision, to keep his eyes steady. It has to do with the deviating movements of the eyes which are present from birth. We find it in cases of corneal opacity from ophthalmia neonatorum, in congenital or very early cataract, in early irido-choroiditis, in coloboma, and in albinism. Not very rarely no cause can be discovered.

The acquired variety has generally some central cause. It occurs, for example, in some cases of meningitis, hydrocephalus, and encephalitis lethargica, in Friedreich's ataxia, and occasionally in intracranial tumours. Often, however, no local or general cause can be found.

The peculiar **nystagmus of spasmus nutans** is a co-ordination neurosis and has to do with the acquired movements of convergence (p. 717). It differs from the ordinary kind not only in its being usually (though not always) associated with head movements, but also in the following particulars¹:—

(a) *Its Time of Onset.*—It is always acquired—setting in, generally, between the ages of six and twelve months while the baby is learning to control his ocular movements. It seems never to occur except in early childhood.

(b) *Its Tendency to Recovery.*—It is always recovered from within a few weeks or months, although relapses may occur. Ordinary conjugate nystagmus is generally permanent.

¹ J. Thomson, *Brit. Med. Journ.*, 1901, i., 763.

(c) *The Character of the Movements.*—Ordinary nystagmus is nearly always bilateral and horizontal. This form is often unilateral, often vertical or rotatory, and not rarely shows different directions of movement in the two eyes—*e.g.*, horizontal or vertical in one, and rotatory in the other.

Ordinary horizontal nystagmus is *always* conjugate—the antero-posterior axes of the two eyes remaining parallel to one another all the time. The nystagmus of spasmus nutans, when bilateral and horizontal, is usually convergent—that is to say, the corneæ incline alternately towards and away from one another. Rarely it also is distinctly conjugate. In some cases the nystagmus is so fine that it is almost or quite impossible to make sure of its type.

In those cases of spasmus nutans in which the nystagmus is rotatory, the movement is not a simple rotation of the globe round its antero-posterior axis, but a sort of irregular circumduction—the central point of the cornea passing through an ellipse or some such rounded figure.

The Marcus Gunn or Jaw-Winking Phenomenon.

This is an interesting developmental and occasionally hereditary rhythmical contraction of the upper eyelids during certain movements of the jaw. At times it is associated with lateral movements of the jaw, and at other times with clenching of the jaw. It is usually present in only one eye but on rare occasions it has been observed in both eyes. Fig. 214, from a cinema film of an example in a child aged fourteen months, illustrates the condition very well.¹ The exact cause is disputed, but Bishop Harman² has suggested that it is due to a reversion to a more primitive state.

The Fundus Oculi.

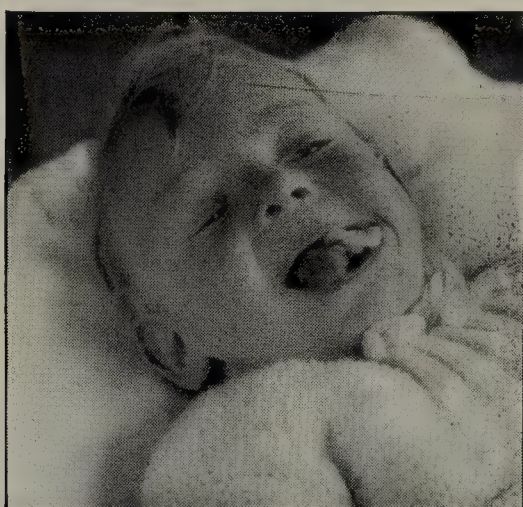
Examination of the fundus oculi is of considerable importance in a variety of pathological states and should never be omitted. Nowadays the electric ophthalmoscope makes such a procedure comparatively easy even in the youngest infant.

¹ L. Findlay, *Proceedings of the Royal Society of Medicine*, August 1931, xxiv. (Section for the Study of Disease in Children, p. 65).

² N. Bishop Harman, *Trans. Ophth. Soc. U.K.*, 1903, xxiii., 356.



A



B



C



D



E



F

FIG. 214.—Selected from cinema film showing phases of Jaw-Winking Phenomenon. Note absence of contraction when child is at rest (A), and when the mouth is open (B and C); but marked contraction of levator palpebrae superioris of left eye during biting movement (D, E, F).

The presence of patches of choroidal atrophy, round which the pigment is usually concentrated, is characteristic of lues, but an increased granularity of the choroidal pigment (pepper-ground retina) is also suspicious of syphilitic infection. Very occasionally isolated patches of choroidal atrophy, usually limited to one eye, may be the result of previous tuberculous infection.

Miliary tubercles, which appear as pale, raised, circular or oval areas along the course of the vessels, are occasionally found, and should always be searched for in cases of suspected miliary tuberculosis, with or without meningitis.

Optic neuritis is specially characteristic of cerebral tumour, but it may also be present in meningitis if this is of some duration; hence it is more frequently found in the later stages of tuberculous than meningococcal or other types of the disease. Optic neuritis indistinguishable from that present in cerebral tumour may, but only very rarely, occur in nephritis in childhood.

Optic atrophy is found in many examples of mental deficiency and hydrocephalus. Optic nerve atrophy may occur as a hereditary and familial condition (*Leber's Disease*); though it does not as a rule develop till after puberty (18 to 23 years),¹ it may declare itself during childhood (7 to 8 years).²

Hæmorrhages into the retina are characteristic of birth cerebral injury, pachymeningitis hæmorrhagica, purpura, and the severe blood diseases, *e.g.*, leukæmia.

Cervical Opisthotonos.

Head retraction with rigidity of the neck generally indicates the presence of meningitis. When it occurs in the tuberculous form it is often slight and intermittent; but in meningococcal and other septic varieties, in which it is almost always present, it is usually an early symptom, is very severe, and persists till death; or, in cases which recover, for many weeks. Cervical rigidity also sometimes occurs in chronic hydrocephalus (Fig. 215), cerebral tumour, and poliomyelitis.

Although head retraction when severe in degree is usually a symptom of intracranial disease, it is sometimes met with under other conditions. We may find it in acute otitis media,

¹ Th. Leber, *Arch. f. Ophth.*, 1871, xvii., ii., 249.

² W. F. Norris, *Trans. Amer. Ophth. Soc.*, 1880-84, iii., 673.

in tetanus, and in typhoid, diphtheria, and pneumonia, without any brain lesion. Not very rarely it occurs in wasted babies with dyspepsia,¹ and a minor degree of it is also sometimes seen in tetany. It is important to remember that a certain degree of nuchal rigidity is present after lumbar puncture done for whatever cause.

Various surgical affections, such as acute retropharyngeal adenitis or abscess, and cervical caries, cause stiffness of the neck with drawing back of the head, which may be mistaken for cervical opisthotonos of intracranial origin.

The Superficial Reflexes.

In early childhood the skin reflexes are generally poorly marked and they are never exaggerated. In young babies some of them, such as the abdominal, are often absent altogether. Sometimes, however, as R. Laurent² has pointed out, during the first nine months the area, stimulation of which elicits the abdominal reflex, is considerably enlarged. This is



FIG. 215.—Chronic Hydrocephalus following Meningitis, showing head retraction. (Girl of 1 year.)

never found after the fourteenth month. The cremasteric reflex is often absent in young children.

The Plantar Reflex.

As Babinski stated in his first paper on this subject, an extensor response is normally present in new-born children. This is not, however, the only peculiarity of the plantar reflex in infancy. The effect of stimulating the sole in a young baby has been described as follows by James Collier³: "The

¹ Beattie and Selby, *Edin. Hosp. Rep.*, 1896, iv., 343.

² "Evolution des Réflexes chez l'Enfant," *Thèse de Toulouse*, 1905.

³ *Brain*, Spring, 1899, 78.

earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes with eversion of the foot and dorsiflexion of the ankle, and subsequently flexion of the hip and knee. Strong stimulation causes a general irregular movement of the limbs and trunk." This "infantile response" "contrasts most markedly with the adult form, with the contracted flexed toes, inverted foot, and the early hip response."

As the child grows older, the preponderance of dorsal over plantar flexion steadily diminishes, until by the end of the first year the proportion of each is about 50 per cent. of the whole.¹ During the second year plantar flexion becomes increasingly common. About this age, however, the reflex is often not easily obtainable. By the third year a flexor response is practically always present in healthy children.

Although the transition from dorsal to plantar flexion normally occurs about the time when the child is learning to walk, it does not seem to be due to this; for plantar flexion is found in many children who cannot walk, and is delayed in some who do. In weakly, backward children the appearance of the normal flexor response is apt to be delayed, while in strong, vigorous infants it appears sooner than usual.²

During sleep the plantar reflex is less definite, but generally its type is the same as when the child is awake. Occasionally, however, children are met with, even as old as twelve, who present a typical infantile response during deep sleep, although at other times they show the ordinary plantar flexion. The same thing may sometimes be noticed during chloroform narcosis and immediately after an epileptic seizure.

In older children the significance of changes in the plantar reflex is the same as in adults. In infants under two, Babinski's symptom has generally no value as a sign of disease. If, however, a flexor response is obtained in premature and ill-developed infants who are late of walking, it is a good omen.³ It shows that the cord is healthy, and therefore justifies the hope that the children will ultimately be able to walk.

¹ Engstler, *Wien. klin. Wochenschr.*, 1st June 1905, 567.

² Passini, *Wien. klin. Wochenschr.*, 1900, No. 41.

³ Léri, *Revue neurolog.*, 30th July 1903, 689.

The Lip Reflex of New-born Children.¹

When we consider that the act of sucking is the most fully developed co-ordinated act of which the newly-born child is capable, it is not surprising that the lips should be the seat of a special reflex at birth. It is probable that the lip reflex serves a useful purpose in assisting the infant's first unpractised attempts at sucking. By its means the mouth assumes automatically a more convenient shape for receiving and retaining the nipple. In the same way the mother's nipple, when subjected to mechanical stimulation, contracts so as to become thinner, longer, and harder, and therefore more easily grasped and retained by the infant's mouth.

The reflex is best elicited by a series of gentle taps on the upper lip a little above the angle of the mouth, or on the under lip a little below it (Figs. 216 to 218). It may, however, be got by tapping anywhere on the lips in a well-marked case, and sometimes over a considerable part of the cheek. A gentle touch on the lips, such as might be given by the mother's nipple, will also originate the movements.

On tapping the upper lip there is often, first of all, a slight momentary jerk. This is generally towards the side tapped, but sometimes towards the other side. Almost at the same time the lips close, if they have been parted, and become deliberately pursed together so as to pout a little. As the tapping is repeated, the protrusion of the mouth becomes more and more definite (p. 684). In some instances the projection is straight forward, but generally the central point of the mouth turns distinctly towards the side *opposite* to that tapped. Both upper and lower lips participate in the pouting. In some cases the preliminary jerk is not seen. In others, after repeated tapping, there are to-and-fro sucking movements of the tongue along with the pouting. When the lower lip is tapped, the resulting phenomena are much the same.

The lip reflex seems to occur more or less distinctly in all healthy new-born babies when they are sound asleep, and in

¹ See Escherich, *Traité des Maladies de l'Enfance* (Comby et Grancher), Paris, 1898, iv., 755; Loos, *Verhandl. d. Gesellsch. f. Kinderheilk.* (Frankfurt-a.-M., 1896), Wiesbaden, 1897, 1; Thomson, *Review of Neurol. and Psychiatry*, March 1903, i., 145; Toulouse and Vulpas, *Comptes rendus hebdomadaires des séances de la Société de Biologie*, Séance du 11 Juillet 1903, lv., 152.

THE LIP REFLEX.



FIG. 216.—Before tapping.

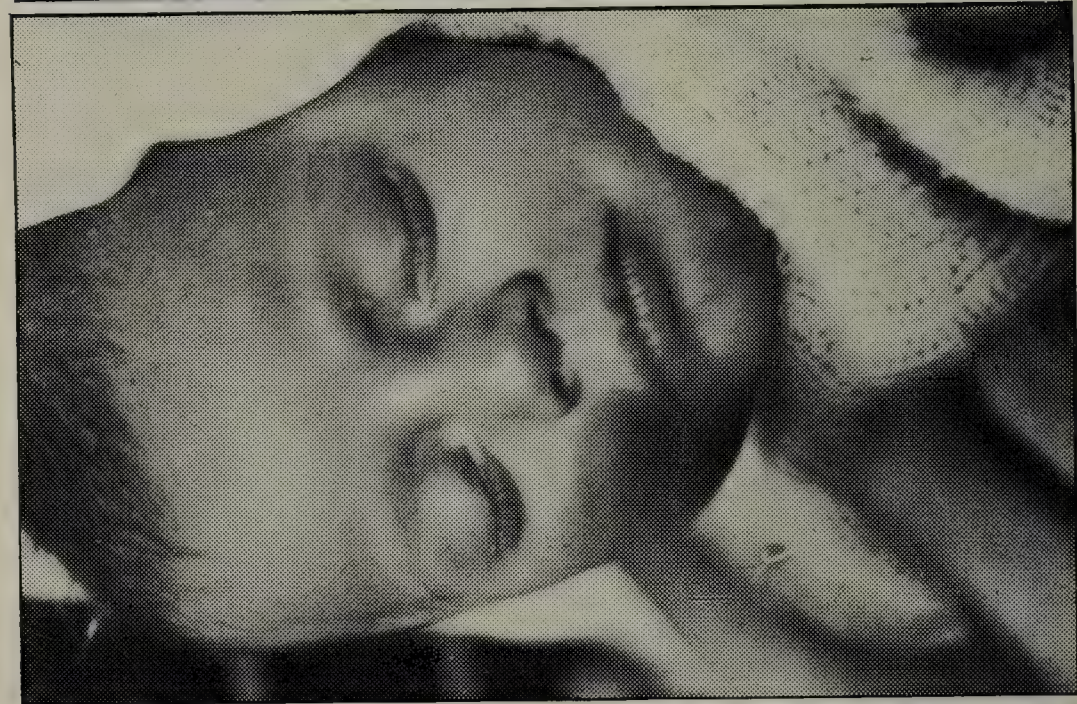


FIG. 217.—After first tap.



FIG. 218.—After several taps.

a considerable proportion of them when they are sleeping lightly, or even are only drowsy. We have seen it in a few new-born infants who were evidently wide awake; but this is rare.

As the children grow older the reflex is less frequently found, and is only present when they are sound asleep. Until the end of the third or fourth year it is fairly common. After that it is less so, and it is usually also less vigorous. We have found it occasionally in older children up to twelve years, but have not examined for it in adolescents or adults. The lip reflex must not be confused with "facial irritability" (Chvostek's phenomenon, p. 539).

Infants of a few months who are taking large doses of chloral on account of repeated convulsions often show a peculiarly strong lip reflex. Probably this may be due partly to their being able to stand more percussion than usual without waking. We have, however, seen it strongly present in several babies who were taking convulsions and who had not had any sedative drugs.

Some cases of spastic diplegia, while awake, show energetic chewing, sucking, and swallowing movements when touched on the lips and other neighbouring parts. This phenomenon seems to represent an exaggeration of the normal lip reflex.¹

Myotatic Irritability.

A high degree of myotatic irritability is often found in children, and occurs very often in cases in which there is no tuberculous disease.

State of the Knee-Jerks.

To obtain the knee-jerks in a young child, while he is sitting on his mother's knee, it is best to place the palm of the left hand under his foot, and to support his leg in this way as by a stirrup; then, on tapping gently on the tendon with the middle finger of the right hand or with a percussion hammer, the amount of movement which takes place is readily felt as well as seen. In strong, healthy children the knee-jerk is generally easily obtained. In those with weak and flabby muscles it may be difficult to make sure of its presence. When difficult

¹ H. Oppenheim, *Monatsschr. f. Psychiat. u. Neurol.*, 1903, xiv., H. 4, 241.

to elicit, it may sometimes be got by gentle tapping with the limb considerably extended.

In healthy infants under six months the knee-jerks are generally brisk, though they may sometimes be difficult to obtain owing to the child's restlessness. Considerable increase of the knee-jerks may be present in nervous children apart from any organic disease.

They are nearly always absent in diphtheritic paralysis, in peripheral neuritis, in progressive muscular atrophy and pseudo-hypertrophic paralysis, in those cases of infantile spinal paralysis in which the extensors of the thigh are affected, and in various other conditions. The knee-jerks are said to be absent in many cases of croupous pneumonia in children (Pfaundler,¹ 27.5 per cent.; Kephallinós,² 41.5 per cent.). In our experience the loss of the knee-jerk occurs so seldom in pneumonia that it is of no value as a diagnostic feature, as has been stated.

The knee-jerks are increased in most cases of infantile cerebral paralysis (though, in some, the extremely spastic condition of all the muscles renders the movements even less than normal); also in myelitis and pressure on the cord above the lumbar region, and in many other forms of organic disease of the brain and spinal cord. They are also exaggerated, though to a less extent, in children who have suffered from a prolonged feverish illness such as enteric fever, and in severe and long-standing hysteria.

Kernig's Sign.

This consists in a reflex contraction of the hamstring muscles which occurs whenever an attempt is made to extend the knee-joint while the thigh is kept at right angles to the trunk. It can be elicited either when the patient is sitting on the edge of the bed or when he is lying on his back.

When other symptoms of meningitis are present, the discovery of Kernig's sign is a further confirmation of the presence of that disease. The importance of the sign in clinical work is, however, difficult to estimate. It is commonly present in cerebro-spinal meningitis, though in some typical cases it is absent throughout. It is not rare in tuberculous

¹ *Münch. med. Wochenschr.*, 1902, xlix., No. 29, 1211.

² *Ibid.*, 24th July 1906, 1460.

meningitis, although less common in it than in the meningococcal form. It may also be found in many conditions in which the meninges are not inflamed; for example, in intracranial hæmorrhage and abscess, in acute poliomyelitis, in some cases of pneumonia, uræmia and typhoid fever; and in other intestinal diseases with nervous symptoms (meningism).

In young infants, owing to the tendency to hypertonicity of the muscles, Kernig's sign often cannot be satisfactorily demonstrated.

Brudzinski's Neck Sign.¹

To elicit this sign, the child is laid flat on his back and his head is rapidly bent forward by the observer's left hand, which is placed on the occiput, the chest being held down by the right hand. In the great majority of cases of meningitis, a marked flexion of the lower limbs at the knee and hip-joints occurs when the head is brought forward. This sign is said never to occur in healthy children or in those who are suffering from other than meningeal disease. It occurs with equal frequency in all varieties of meningitis.

Tache Cérébrale.

This sign—the unusual persistence of marked hyperæmia in the track of a light scratch on the skin—is common in the later stages of severe cerebral cases, but is of little diagnostic value, as it may occur in enteric and other fevers without any brain lesion being present.

Paralysis.

In investigating any apparent loss of muscular power in a child, we have, *first*, to decide whether it is a true paralysis, or merely a pseudo-paralysis resulting from pain on movement, from extreme flabbiness of the muscular tissue, or from some malformation; and *second*, if a true paralysis, whether the lesion is in the brain, cord, or peripheral structures, or is a functional palsy such as may be met with in hysteria, or as the result of peripheral irritation.

¹ Brudzinski, *Archives de Méd. des Enfants*, 1909, xii., 745; Morse, *Trans. Amer. Pediat. Soc.*, 1910, xxii., 50; Northrup, *Journ. Amer. Med. Assoc.*, 1911, lvi., 114.

Pseudo-Paralysis.

Disinclination to move the limbs, owing to the pain which movement causes, is seen in cases of *disease* or *injury* of the muscles, ligaments, and bones. If present in the upper arm soon after birth, it may be due to injury of the upper epiphysis of the humerus during delivery. In syphilitic infants between four and twelve weeks old it is generally caused by *specific osteo-chondritis*, and most frequently affects the upper limbs. A similar pseudo-paralysis occurs in cases of *infantile scurvy*, but it is oftenest met with in them between eight and fourteen months old, and affects the lower more often than the upper extremities. True paralysis (as from anterior poliomyelitis or peripheral neuritis) is frequently accompanied at first by severe pain on movement.

The pseudo-paralysis seen in *rickets* seems to be due more to muscular debility and laxity of the ligaments than to tenderness of the structures, although that has probably some share in producing it. While it may be noticed during any stage of rickets, it rarely causes difficulty of diagnosis except late in the course of the disease, when the other characteristically rickety symptoms have more or less subsided. It is always symmetrical, and is generally noticed in the lower limbs, although the upper limbs if examined will be found similarly affected.

The weakness caused by *congenital dislocation of the hip-joint*, and by various other congenital malformations of the limbs, is apt to be mistaken for infantile paralysis.

Tremor.

Tremor is rather a rare symptom in young children. A general coarse tremor is sometimes met with, however, in certain of the infectious diseases such as enteric fever and influenza, and in tuberculous meningitis. Local tremor of a limb is seen in some cases of cerebral and cerebellar tumour.

Ataxia.

A transient form of ataxia is sometimes seen in children with a normal nervous system who have been confined to bed for some weeks with any acute feverish disease such as scarlet

fever, and also in those who have been kept lying constantly for months owing to spinal caries. Generally, however, ataxia is a symptom of more or less grave significance. It is characteristic of such conditions as tumours of the cerebellum and mid-brain, of Friedreich's disease, of chorea, of some cases of diphtheritic paralysis, and of certain rare cases of hysteria.

Three varieties of cerebellar diplegia in children, of which the main symptom is ataxia,¹ have been described. These are: (a) *Congenital Cerebellar Ataxia*—i.e., congenital ataxia in mentally normal children, with practically no other indication of intracranial disease; this has a tendency to steady improvement, and sometimes almost complete recovery occurs in the course of development. (b) *Acute Ataxia* (encephalitis cerebelli)—these cases develop suddenly in healthy children with acute symptoms. They tend to improve, and frequently recover. Most of these cases set in after one of the infectious diseases. When recovery takes place, it may be complete in one, two, three, or more months (Griffith).^{2, 3} (c) *Progressive Cerebellar Ataxia*—this develops slowly in previously healthy children and has a tendency to progressive increase.

Sensory Defects.

Defects of ordinary sensibility are less frequently met with in children than in adults; and, unless very decided, are difficult to estimate. General tenderness to touch and movement usually arises from the presence of some such general disease as rickets or scurvy, of a local injury of the part, or, less frequently, from lesions of the nervous system, such as basic meningitis or acute poliomyelitis. Local areas of tenderness of the skin or subcutaneous tissue are not common in children, but, when present, they are very useful in drawing attention to the presence and situation of visceral disease.

Electrical Reactions.

When it is important to ascertain the electrical reactions in a young child, it is generally best to administer chloroform,

¹ F. E. Batten, "Ataxia in Childhood," *Brain*, Autumn and Winter No., 1905, cxi. and cxii., 484.

² J. P. Crozier Griffith, "Acute Cerebellar Ataxia in Children," *Amer. Journ. of Med. Sci.*, Jan. 1916, cli., 24.

³ S. Graham, *Arch. Dis. Child.*, 1930, v., 146.

as otherwise the child's struggles will make a proper examination exceedingly difficult, if not impossible.

Lumbar and Cistern Puncture.

Lumbar puncture is a simple proceeding, and with ordinary aseptic precautions it is quite safe. It does not demand an anæsthetic, but in infants and in delirious or nervous children this is advisable. In the young infant, owing to the smallness of the spinal canal, the least movement not only makes the operation difficult but apt to be accompanied by bleeding, and in the delirious patient an excessive amount of force is necessary to restrain any movements.

In carrying out the operation the child is laid on his side with his back bent forward as much as possible. A line drawn across the spine at the level of the upper border of the iliac crests will then be found to pass over the fourth lumbar spine. Just below it, in the fourth space, is the best site for the puncture. An ordinary exploring needle or fine trocar is passed into the interspace in the middle line, to the depth (in a child) of $1\frac{3}{4}$ to 3 cm. Not more than half an ounce or an ounce should be removed, even when the fluid is plentiful, except in special chronic cases. Should the fluid not flow easily, a stilette may be passed, but suction by means of a syringe should only be very carefully employed.

When fluid cannot be obtained by the lumbar route, **cistern puncture**, *i.e.*, tapping the *cisterna magna*, should be practised. This operation is also necessary for the introduction of lipiodol to delimit the upper level of any suspected obstruction, and it is claimed to be the best site for the introduction of sera. It is, however, contraindicated in the case of a cerebral tumour. To tap the cistern the patient is laid on his side with the head slightly flexed and resting on a sand-bag, so that the spinal column is horizontal. The nape of the neck is prepared in the usual way, the occipital tubercle and spine of the axis are identified, and the needle (as used in lumbar puncture) inserted midway between these two points and pushed in an upward and forward direction in line with the external auditory meatus. At a depth of 1.5 to 2 cm. the occipito-atlantoid ligament will be encountered just before entering the cistern. If the needle impinges on the occiput it should be withdrawn a little, the point slightly lowered, and again pushed forward at the new level.

After both lumbar and cistern puncture the patient should always be kept lying for twenty-four or forty-eight hours.

Normally cerebro-spinal fluid is crystal-clear and colourless, and comes through the needle slowly, drop by drop. In pathological conditions it is often turbid, blood-stained, or of a yellowish colour (xanthochromia), and the tension is sometimes so great that it spurts out. The amount of fluid is usually increased in meningitis of any kind, in intracranial tumours, in uræmia, and in some cases of obstructive hydrocephalus. It is blood-stained in hæmorrhage on the cerebral surface, xanthochromatic in old hæmorrhage and in spinal compression, and turbid, even amounting to pure pus, in meningitis. The cell content, the protein content and chloride content, as well as the presence or otherwise of organisms as evidenced in film and by culture, must invariably be investigated, as it is in this way alone that not only the existence of, but also the special type of a disease can be recognised. The findings in the different pathological states are given in Table on page 776.

As a form of treatment, lumbar or cistern puncture is often disappointing. The relief of tension afforded by it in tuberculous meningitis generally leads to no corresponding improvement in the symptoms. Sometimes, however, striking relief follows it in purulent meningitis, and, if so, its repetition may be beneficial. In serous meningitis marked relief is invariably obtained. It has also proved useful in convulsions, in uræmic and other forms of severe headache, in some cases of vertigo, in some of head retraction without meningitis, and in certain forms of chronic hydrocephalus. When hydrocephalus is accompanied by convulsions and screaming attacks, these may often be stopped by periodic withdrawal of a small quantity of cerebro-spinal fluid.

The indications for and technique of **ventricular puncture** are given on p. 787.

CHAPTER XXVIII

FUNCTIONAL NERVOUS DISORDERS

Disorders of Sleep

THE characters of normal sleep and its great importance for the child's health have been already discussed (Chapter II). We have now to consider how it is changed in sickness, and the effect on the general health caused by scanty or otherwise abnormal sleep. A child sometimes sleeps too much; far oftener, not long enough. His sleep may be disturbed and lacking in the peacefulness which should characterise it in health; and at times it may be interrupted by nightmare or night-terrors.

Excessive sleepiness may arise from functional disturbance or organic disease of the stomach, bowel, or liver, or from the action of drugs; but, in most cases in which it occurs to an extreme degree, it is a symptom of intracranial disease (see Drowsiness, p. 663).

Sleeplessness.—In children, as in adults, sleeplessness may depend either (*a*) on bodily, or (*b*) on mental causes.

(*a*) *Bodily Causes.*—In young infants, wakefulness most commonly indicates either hunger, thirst, or indigestion, and calls for a careful revision of the feeding. Occasionally it is due to cold from inadequate coverings but, on the other hand, it may be due to an excess of bed-clothes. Disturbed sleep is, moreover, a common manifestation of many morbid conditions. It is characteristic of rickets and congenital syphilis, of coryza, constipation, phimosis, and other ailments that give rise to discomfort, of eczema and other sources of itching, of otitis media, difficult dentition, and other painful affections. It is also often seen in pneumonia and toxæmia of various other kinds. Defective ventilation is another common cause; many babies who are wakeful in bed, sleep soundly in their perambulators in the open air; and a widely open window in

the sick room often proves an excellent soporific. Sleeplessness is also due, at times, to the child being put to bed in a brightly lighted room with adults talking beside him.

In older children sleep is frequently disturbed temporarily as the result of indigestion and late meals, and of such local causes of discomfort as adenoids and cold feet. Difficulty in falling asleep at night is often a sign that the school work is proving too much for the child's strength. Occasionally the nights are disturbed on account of a constantly recurring cough.

Extremely severe and intractable insomnia, lasting for months or years, is one of the most characteristic sequels of encephalitis lethargica. A much less severe degree of it is often a troublesome symptom in pneumonia and in toxæmia of various kinds.

(b) *Mental Causes*.—Even in young infants, habitual wakefulness may be largely a matter of bad training and excitement. Not infrequently a troublesome baby, in the charge of a restless and vivacious woman, will become a good sleeper soon after he is taken over by a stolid, restful nurse.

In little children, going to sleep, like going to stool and other good habits, is largely assisted by suggestion. When in the quiet routine of the nursery his bedtime comes, drowsiness is unconsciously suggested to the baby as he sees his cot ready and preparations being made for the night. He sees that those round him expect him to go off to sleep and he soon acquiesces. His mother's cradle song, and the common (though unnecessary and undesirable) habit of rocking, probably owe much of their efficacy to this sort of suggestion.

In those children, however, who are neurotic or have been spoiled, this natural process is easily upset by an attack of temper or some undue excitement, as well as by bodily causes. When this occurs, it may be very difficult to restore the baby's equanimity. Scolding and petting are of no use and usually make matters worse. They fix the child's attention still more on himself, and on succeeding nights he is sure to remember the conflict, and will probably renew it. For, though the struggle was in itself unpleasant, it was accompanied by the gratifying feeling of having aroused a great sensation among his seniors, and having got his own way.

In cases of this kind, the recovery of the natural inclination

to go to sleep depends largely on how far the mother and nurse are able to supply the necessary suggestion ; in other words, to make the baby feel that he is expected to go to sleep, and that no fuss at all will be made if he is long about it.

Occasionally, sleeplessness in children of two years or over is due to the presence of morbid fears which are aggravated by the dark. Owing to a child's reserve, the cause in such cases may be very difficult to discover.

In older children sleep is often disturbed by an undue amount of mental exertion or emotional excitement shortly before going to bed.

The main thing in the management of sleeplessness is the discovery and treatment of its cause. When it is due to pain in acute disease, as at the onset of croupous pneumonia or pleurisy in a strong child, an opiate such as Dover's powder may be beneficial. If it occurs under conditions of exhaustion, it is to be treated by warmth and stimulants. If there is abdominal uneasiness, a large poultice is often a good thing, or an enema of hot water, or a dose of castor oil. Antipyrine is useful when the sleeplessness is due to itching, and in the temporary restlessness of an acute feverish illness. In habitual wakefulness, however, whatever its cause, drug treatment is almost always bad treatment. The symptom is a danger-signal ; and its cause, whether it consists in bad training, in bad habits of life, or in the persistence of some local irritation, must be found out and removed.

Nightmare and Night-Terrors (*Pavor nocturnus*).—These are commonly met with in children between two and eight, and are rare after twelve years old. Some authorities, as Coutts,¹ regard them as essentially different. Others, as L. Guthrie,² see no clear line of distinction between them as to nature and significance. In *nightmare*, the child wakes suddenly from a somewhat disturbed sleep with a loud scream. Usually he cannot tell what is frightening him ; and, though he is fully conscious of his surroundings, it is some time before he can be induced to fall asleep again. A second attack may occur in the same night. In *night-terrors* proper, according to Coutts, the sleep from which the patient awakes has usually

¹ *Amer. Journ. of Med. Sci.*, 1896, cxi., 156, and *Encyclop. Medica*, viii., 360.

² *Allbutt and Rolleston's Syst. of Med.*, 1910, viii., 804.

been peaceful and his screams are due to his having "seen visions" rather than "dreamed dreams." Thus he cries out that a big dog or a black man is threatening him; or he mistakes his mother, standing at his bedside, for a horse about to run over him. Night-terrors are a less common and also a less trivial symptom than nightmare; and there is rarely a second attack in the same night. In both, the attack generally sets in shortly after the child falls asleep—at the very time when, as A. Czerny¹ has shown, sleep is deepest.

There are generally various elements in the *causation* of these conditions, and the share of each is difficult to apportion and to express. The general state of the child's nervous system is important as well as his age. Children of nervous families have these attacks oftener and more severely than others. The special susceptibility of the highly-strung, imaginative, and often precocious child should always be borne in mind. Some of the worst instances of night-terrors we have seen began immediately after a severe fright. Local sources of discomfort, such as enlarged tonsils, adenoids, and chronic indigestion, are often partly to blame. A late and heavy meal is, of course, a recognised cause of nightmare.

The *treatment* of nightmare and night-terrors consists in attention to any predisposing and exciting causes which can be found. If adenoids or enlarged tonsils are present, the symptoms are often permanently cured by their removal. In many cases, whether adenoids are present or not, attention to the diet will speedily remove the nervous symptoms. The restriction of starchy foods, vegetables, fruits, and sweets, combined with the administration of 6 or 8 gr. of bicarbonate of soda, an hour before each meal, often works wonders when the digestion is deranged. Regular night-terrors can sometimes be rapidly stopped by a sufficient dose of antipyrine or some other sedative given at bedtime; and the strengthening of the nervous tone by the administration of a carefully given cold douche at bedtime has sometimes the best results.

It is, however, most important always to remember that the occurrence of such attacks usually demands an investigation of, and probably a change in, the child's habits and surroundings as to diet, education, amusements, and hygiene. It may be due

¹ *Jahrb. f. Kinderheilk.*, 1892, xxxiii., H. I., 1.

to the child's individual make-up, when, of course, treatment is not only less called for but is also less successful.

Somnambulism.—Somnambulism does not usually occur in children under ten, and it is met with mostly between that age and adolescence. Its significance and treatment are the same as in night-terrors.

Day-Terrors.—Attacks resembling night-terrors are occasionally met with during the daytime. Their causation, significance and treatment are similar to those of the night seizures. They constitute an even more urgent sign of a vulnerable nervous system.

Other Morbid Fears in Little Children.—It is not uncommon for little children to have paroxysms of fear when they suddenly encounter a strange animal, a grotesque toy, or a member of one of the classes, such as policemen and "black men," that have a bad reputation in the nursery. Such incidents are not abnormal and scarcely lie within the domain of the doctor.

Occasionally, however, we are consulted about fits of terror which are distinctly morbid, not only in their severity, but also in the fact that they are set up by objects which the child has hitherto regarded with a friendly interest and no anxiety. Evidently some idea has got into his head which has altogether changed his relation to them. One has known attacks of this sort brought on by the sight, even at a distance, of such objects as a puppy, a motor car, an airship, or a pair of crutches; and every subsequent appearance of the object in question is followed by a paroxysm of screaming.

These attacks may conveniently, as far as treatment is concerned, be divided into two classes according to the degree of their severity—severe paroxysms in which the child is beside himself with terror and loses all his self-control, and slighter attacks in which, though intensely anxious, he can still control himself to some extent by an effort of will. These two are obviously of the same nature and due to the same causes.

Treatment.—The first thing to do in a case of this kind is to set the parents' minds at rest by assuring them that the attacks, though they must not be allowed to go on, are not dangerous in themselves, if judiciously dealt with; and that their occurrence need cause no anxiety as to the child's mental condition or his future character. Fear does not necessarily signify cowardice and, though his terror shows that the child

has a vivid imagination, it does not prove either delicacy or timidity. The patient indeed is often noticeably brave about other things that children are afraid of.

The measures to be taken with regard to the attacks vary according to the degree of the terror, and the extent to which his power of self-control is affected. In the case of the severe paroxysms, the main thing is to prevent him, for a considerable time, from seeing the objects which frighten him, and from hearing them spoken about. Once the paroxysm of fear has seized him he cannot help himself, and the only thing for the mother or nurse to do is to take him in her arms and give him all the sympathy and comfort possible till he can be got to forget his troubles in sleep.

The lesser attacks are also to be treated with the greatest sympathy; for, to the child, the causes of his fright are painfully real; and anything in the way of punishment, reproof, ridicule, or making light of his symptoms, is altogether out of place and can only do harm. As he has not lost all self-control, a good deal can often be done to help him to pull himself together when he feels the sensation of fright coming on. Two things may be mentioned that will assist him in doing so. The first of these is a cheerful and confident demeanour on the part of his seniors. This will go a long way towards enabling him to control himself. Fear, and the anxiety which predisposes to fear, are notoriously infectious; but calmness and confidence are equally catching. The other way in which the parents may help the child is by anticipating the onset of his fear, and suggesting to him at once to go and do something that will turn his thoughts in another direction. For example, should a visitor be announced whose appearance is likely to recall the dreaded subject, the chance of a screaming attack will be greatly lessened if the child is sent quickly to fetch her a chair or a flower, or to do some other easily performed service that takes him out of himself for the time.

In the earlier and more severe stages of the trouble, the child's fears should never be alluded to in his presence. Later, however, when he is beginning to recover and to regard the causes of his fear more normally, it will be well to introduce the subjects occasionally, to speak casually of their good points and harmlessness, and perhaps also of himself as being now too big and sensible a boy to be afraid of such things.

Insanity.

Ordinary insanity is rare in childhood and is scarcely ever recognised in infancy. When it occurs in early life there is usually a strong neurotic taint, and sometimes also a special exciting cause. Often the patient himself has already shown other signs of a weak nervous system; mentally defective children not uncommonly become insane.

In the rare cases in which *mania* occurs it is sometimes associated with hysteria and sometimes with chorea. Occasionally it is due to septic poisoning, such as arises from a focus of diseased bone. When this is so, the mental symptoms are at once relieved by removal of the source of the sepsis. Most of the young children that we have seen with maniacal symptoms were epileptics, and many epileptic children show a high degree of mental exaltation and irresponsibility between their attacks. In epidemic encephalitis in children a condition closely resembling mania is not uncommon, and is of very bad prognostic significance.

Melancholia occasionally occurs in childhood after such exhausting complaints as influenza and enteric. Mental depression is also not uncommon in badly-nourished children in the course of various bodily diseases. Abnormally low spirits have a bad prognostic significance when they occur in a chronic ailment such as valvular heart disease.

A **temporary loss of moral control** which is akin to melancholia is occasionally seen in children of school age. It is important that such cases should not be mistaken for instances of permanent mental defect, and that prompt and sensible measures of treatment should be taken. The patient, who usually comes of a neurotic stock, and has probably been subjected to a great nervous strain—from bullying or some other form of unsympathetic treatment, or perhaps from an exhausting bodily disease—becomes quite changed in character and habits. He shows a lack of concentration in his school work, and, for the first time, is heedless, dirty, and untidy in his ways. He is untruthful, and shows no sign of shame when found out, though morbidly afraid of punishment. He is dull and unhappy, wanders about aimlessly, and talks aloud to himself. Punishments only make him more miserable.

In such cases immediate and energetic *treatment* is called

for. The bodily health must, of course, be seen to; but the main thing is a complete change of the child's surroundings. His ordinary lessons must be stopped, but it is advisable that a full routine of simple, pleasant duties should be instituted, preferably out of doors, and that he should have no time for loafing. He must be treated with the greatest kindness and encouragement, and every effort made to restore his self-respect and confidence.

With proper treatment, the *prognosis* is quite favourable. Such an attack, however, is a danger-signal, and is not to be forgotten in planning the boy's future life.

Various kinds of **temporary mental aberration** are occasionally met with in growing school children. For example, a girl approaching puberty who has hitherto seemed well brought up, becomes suddenly untruthful and gives long circumstantial accounts of adventures which have no foundation in fact, or develops a practice of indiscriminate pilfering. Such misbehaviour is not to be taken too seriously, and the child should not be spoken to much about it, for the condition is allied to hysteria. If she is treated with patience and a judicious absence of fussing, she will probably soon return to normal ways; but, of course, she should be kept under observation.

Abnormal Naughtiness.—Occasionally, a child of perhaps three or four years, or older, is brought to the doctor because he is becoming so persistently and enterprisingly naughty that his parents have begun to doubt his sanity. He is generally found to be healthy and attractive-looking, and above the average in intelligence, although we are given such a bad account of his conduct. We are told that the child is constantly doing wilful mischief—throwing china out of the window, knocking over flower-glasses, scattering beads or other things that are troublesome to pick up, breaking in on other children's games so as to spoil their play, locking doors and throwing away the keys, pulling out drawers and emptying their contents on the floor, setting fire to things, playing unkind tricks on the cat, or even upsetting the baby out of the cradle. The account of the child's misdoings is rather amusing; and one notices that, in spite of the anxiety which has occasioned the consultation, the parents describe them with just a touch of pride and a feeling that not every child would have the energy and cleverness to be so entertainingly naughty.

When we inquire into the treatment that has been tried, we find that, till recently, the child's mischief has merely been laughed at. Since the parents have thought more seriously about it, however, the young culprit has been subjected to a course of severe corporal punishments, reprimands, and expositions; and it has been attempted also to make him ashamed of himself by repeatedly relating his misdeeds to friends and neighbours, in his presence, with severe comments. These measures have not caused the slightest improvement.

On considering the child's conduct, we see that the things he has been doing were calculated to give him little or no pleasure in themselves; yet they were a real source of gratification because of the strong sensation they produced among his seniors. The effect of his misdeeds in disturbing his parents is the true secret of their attractiveness to him; and so great is the child's enjoyment of this that the beatings and other unpleasant experiences that followed have counted for nothing against it. They have been taken, indeed, merely as further evidence of the greatness of the impression he has succeeded in producing.

What is wrong with the child in such cases is a morbid exaggeration and perversion of the natural desire which all children feel for notice by their elders. He craves to be the most noticed person in the household; and the obvious, though unexpressed, admiration provoked by his boldness and resource is all the approval he needs to encourage him to continue.

This kind of naughtiness differs essentially from so-called "moral imbecility" in the patients being mentally normal and in their being entirely reformed by judicious management.

The proper lines of treatment are clear enough; and, when carried out consistently and thoroughly, they are rapidly and completely successful. They may be summarised as follows:—

(1) All severe corporal punishment must be stopped. It never does any good.

(2) Nobody must ever appear shocked, amused, or even surprised, at anything the child does.

(3) His misdeeds are never to be alluded to, much less described, in his presence; but he should be noticed and encouraged in every way when he is good, and simply ignored when he is naughty. To ignore a child is the greatest punishment that can be inflicted.

Cases of this sort of abnormal naughtiness are met with not very rarely in children who are mentally somewhat defective. In them home treatment is extremely difficult if not impossible ; but the routine of a well-ordered institution is beneficial and soon inclines them to mend their ways.

Certain "Bad Habits."

No consideration of the nervous and mental derangements of infancy would be complete which omitted the curious group of minor psychoses which, for want of a better name, are usually called "bad habits." This interesting group includes such tricks as pica or dirt-eating, sucking the thumb, tongue, etc., biting the nails, head-rolling, head-banging, rocking and swaying movements of the body, and masturbation.

All these habits consist in a morbid exaggeration of some insignificant normal action. The normal act causes only a little temporary satisfaction, while its morbid counterpart often has an extraordinary fascination for the children who practise it. It may not, perhaps, be justifiable to say simply that these habits are infantile hysteria. They certainly, however, occupy among the diseases of infancy a very similar position to that held by hysterical affections among those of later life. They are commonly met with, and are apt to be especially persistent, in children with other neurotic manifestations.

The essential character which serves clearly to distinguish them from other motor neuroses, such as spasmus nutans, chorea, and habit-spasm, which some of them superficially resemble, is their *deliberateness*. The child's will is implicated and what he does is done intentionally, at first at least, because he likes doing it. They have a strong tendency to occur when he is feeling dull and not interested by his surroundings. They are almost always stopped when his attention is taken up with anything else that gives him pleasure.

Habits of this kind often occur during perfect health, but there can be no doubt that many, if not all, of them are specially liable to develop in children whose bodily and mental vigour have been lowered by ill-health, and that they are commonest and most difficult to stop in those who are blind or mentally defective.

Pica or Dirt-Eating.

The children who suffer from pica have a craving to eat such things as earth, gravel, cinders, sand, wall plaster, or paper—sometimes they chew and swallow their own hair, and in rare cases they will even eat fæcal matter. The natural instinct which tells us what is, and what is not, good for food seems absent in them altogether; and the discomfort which must result from the abnormal things which they swallow does not teach them, as it ought, to avoid such things in future. They suffer, as it were, from an hallucination of the appetite. Often there is no ascertainable general or local disease, and the children seem otherwise normal. Sometimes, however, we meet with “cachectic” cases. In these the craving begins with and evidently depends on a diseased condition of the alimentary tract, on the presence of worms, or on anæmia, and it passes off when these are cured. The practice is very common among mentally defective children.

Symptoms.—Pica often begins in infancy (six to eighteen months); as soon as the baby is placed within reach of the things he craves for, he tries to eat them. Thus, even before he can walk, he will be found licking the mud from his father’s boots, or the dirt and gravel from the wheels of his own perambulator. When he begins to walk, he gets access to broken plaster on the walls, and later to all sorts of other things. In the distinctly cachectic cases the symptoms may set in at any age for the first time. In many cases only one kind of unnatural substance is taken; in others a great variety.

If the habit is taken in hand soon after it has begun, it may usually be rapidly checked by the mother or nurse. If it has been allowed to go on for months, however, it may be hard to stop. In the early cases without noticeable cachexia, there is a strong tendency to spontaneous recovery during the third or fourth year, when the range of the child’s interests in life is rapidly widening. Sometimes, however, the habit persists into late childhood or adolescence. Change of scene—for example, admission into a hospital ward—generally stops it at once for the time being. When it does persist, no serious harm follows in most cases. Fatal results, however, sometimes occur from eating sand, gravel, or hair; and severe diarrhœa is not uncommon.

Treatment.—The indications for treatment may be stated as follows¹:—

1. *Keep the child away from the substances* for which he has a morbid craving. All habits are strengthened by practice, and their hold slackens under disuse.

2. *Treat the digestion.* Any local or general uneasiness tends to increase the craving.

3. *Improve the general health, and give iron if the child is anæmic.* These habits have a firm hold on the weakly; the strong more readily throw them off.

4. If possible, *change the child's surroundings* and occupy his mind with new interests. Let him be kept happy and busy.

Wetting the Hands.

Occasionally children are met with who have a constant craving to put their hands into water. This seems to be due to a delusive sensation of dryness, the skin being normally moist. It is a difficult habit to check. We have known it to last for many years in a highly neurotic girl.

Biting the Nails and Fingers.

Traces of *biting the nails* are often seen (Fig. 55, p. 90), and callosities from constantly *biting the hands or fingers* are common, especially in nervous children. In treating nail-biting it is helpful to keep all the nails cut as short as possible; and, in the case of young children, the habit may be discouraged by the local application of a solution of quinine. The most effectual treatment is said to consist in getting a dentist to interfere temporarily with the exact apposition of the incisors.

Sucking of the Thumb or Fingers, the Tongue, or other Parts of the Body, or of other Objects.²

The thumb and fingers are the parts most commonly sucked; less frequently the back of the hand, or part of the arm, or even, in young children, the big toe. The other objects made use of are such things as the mouthpiece of a feeding-bottle, a corner of the sheet or nightdress, or a kneaded-up

¹ J. Thomson, "On Pica or Dirt-Eating," *Edin. Hosp. Rep.*, 1895, iii., 81.

² Lindner, *Jahrb. f. Kinderheilk.*, 1879, xiv., 68; Thomson, *Child-Study Monthly*, Chicago, June 1896, 98.

piece of bread. Generally the same thing is sucked every time, but occasionally the child changes from one thing to another.

The habit is usually "simple," but often it becomes "complicated" in an interesting way. Thus a small boy who is punished for sucking his fingers gets into a way of covering his mouth with his other hand to hide what he is doing; he soon finds an added gratification from this "complication," and always uses it even when alone. Occasionally, also, cases are met with in which the children become violently excited while sucking, and may even injure themselves without seeming to notice it. For example, a patient of Lindner's, while sucking his thumb, used to work so roughly with the little finger of the same hand in one of his nostrils that he made it bleed.

The habit of sucking usually begins in early infancy, but it may start much later. The degree to which it gains a hold over children largely depends on the passive, if not active, encouragement it receives from the nurse or mother. The nurse finds that to check the habit means provoking no end of restlessness, screaming, and ill-temper; while, on the other hand, to encourage it is an easy and sure way of making the child quiet and easily managed—"good," she calls it.

The times when children are most tempted to suck their fingers or other things are shortly before falling asleep, soon after waking, and after their bath; also whenever they are in a low or depressed state of body or mind, or are cold, hungry, or out of sorts. The duration of the habit varies greatly in different cases. In some cases the nurse or mother weans the child from it very soon. In others it is allowed to continue till he goes to school, and then only slowly ceases, owing partly to the notice of his school-fellows.

Sucking usually does little harm unless it is much indulged in or is accompanied by excitement, but it should, practically always, be discouraged. We have, however, seen cases where a baby, with dangerously severe paroxysms of whooping-cough, was more soothed by being taught to suck a "comforter" than by any sedative medicine.

Treatment.—In cases where the sucking is accompanied by excitement, it is important, though sometimes very difficult, to stop the habit. When the finger or thumb is used, this may be best effected by the application of a light anterior splint or a stiff cardboard sleeve to the arm, so that the elbow cannot be

bent. Anointing the part sucked with aloes or quinine may also be helpful. Enclosing the hands in metal gauntlets (aluminium) is an efficacious method of breaking the habit. Generally, if left alone, the habit comes to an end of itself as the child grows older.

Sucking the tongue usually begins in early infancy and is difficult to stop until the child comes to years of discretion. It occurs at one time or another in at least 90 per cent. of mongol children, and in a doubtful case its presence is a point in favour of the diagnosis. It is also not uncommon in cretins and in mentally defective children of other types.

Rhythmical Movements.

Rhythmical movements of various parts are sometimes met with in children. These are of the nature of a bad habit, but are apt to be mistaken for neuroses of another kind.

One of the most striking habits of this class is that described by Gee¹ as *head-banging*. The patient, who is generally a child between two and six years, takes turns of facing his pillow and banging his forehead into it as hard as he can, at regular intervals of a few seconds. This goes on sometimes for several minutes, sometimes for as much as an hour at a time. It may alternate with swaying of the body or head-rolling. It takes place in some cases when the patient is wide awake, and in others when he seems nearly or quite sound asleep. The patients are sometimes deficient in intellect, and sometimes they are cachectic from tuberculosis or otherwise, but often they are quite normal.

Another common habit consists in rhythmical *jerking or rolling the head from side to side* as it lies with the occiput on the pillow. Much less commonly the child may have deliberate *nodding or shaking* movements of the head, while sitting up, which look like an exaggerated and intentional form of spasmus nutans.

Another variety which is often seen consists in a *swaying or rocking* backward and forward of the body. The children sit with a solemn expression, and slowly rock themselves forwards and backwards, sometimes for hours at a time, if left alone. This habit is very common in mentally defective

¹ *St Bart. Hosp. Rep.*, 1886, xxii., 97.

children and specially so in the blind, but it occurs, too, at times in normal children. It may be associated with one or other of the habits above described, or with masturbation; and it is often erroneously regarded as implying the presence of the latter.

Treatment.—A child who begins to practise such a habit should be checked at once, and made to stand up and run about or lie down, whenever he begins it. In normal children who can walk, the habit is not difficult to check in this way.

Masturbation.

Every now and then we are consulted about masturbation in children. The patients may be of any age, but in the majority of cases they are infants. Girls are much more frequently brought to us on this account than boys. This is probably because in them the symptoms are more apt to be misunderstood and attributed to internal distress of some kind, to a fit, or to some other kind of nervous seizure, and therefore allowed to continue.

Symptoms.—The act is practised in various ways—for example, by rubbing the thighs together, by working the body to and fro on the seat on which the child is sitting, or by pressing the vulvar region against the corner of a chair, or, less frequently, by help of the hands. The child seems intensely preoccupied at the time, gets flushed and excited, and often perspires. A sort of panting or grunting expiration usually accompanies the act; and mothers not infrequently refer to this as showing that the child is in pain. If the movements are stopped, however, there are always active signs of annoyance. If the act is not interfered with, more or less exhaustion follows it.

Treatment.—The treatment of confirmed cases of masturbation in *older children*, which have gone on for a long time, is often excessively difficult, and in planning it we have to be largely guided by the circumstances and surroundings of the case. In *young children* who have recently begun the habit the cure is comparatively easy. The masturbation in their case is in no respect a moral offence, and must not be treated as such. At any age the parents' attitude towards it should be firm but altogether unemotional. They should treat it simply as a

gross breach of ordinary good manners. The main indications for treatment are as follows:—

1. *Remove any local irritation present.* Phimosis, preputial inflammation, vulvitis, hyperacidity of the urine, and thread-worms may all be predisposing factors. A mild sedative mixture (ammon. brom. gr. v. to xv. per day) may render the child less irritable and more amenable to control.

2. *Attend to the general health and hygiene.* See that the diet is judicious and not too nitrogenous, and the bed-clothes not too heavy. Order a cold douche in the morning and plenty of open-air exercise.

3. The child *should not be punished* or even scolded; and, once she knows that indulgence in the habit is a direct act of disobedience, that should be the only aspect of the question kept before her. The habit should never be alluded to in her presence and especially never treated as horrible and interesting.

4. The slightest attempt at the act *must be invariably stopped* at once. If this can be done, the child very soon learns that to begin it inevitably means subjecting herself to annoying interference. This spoils the pleasure of the habit altogether and it loses its fascination.

5. If these means fail, *mechanical devices* must be tried to render the act impossible. In cases in which it is always practised with the hands, this can be done by using splints which fix the arms at less than a right angle. If pressing the thighs together is necessary to its performance, it can be stopped by any device which prevents their coming together comfortably. To stop a to-and-fro movement of the pelvis, nothing short of a double long splint, some sort of Phelps' box, or double Thomas's hip-splint, may be sufficient.

6. Much ingenuity should be expended in *keeping the child happy*, busy, and interested in proper subjects.

When such measures as the above are begun early and thoroughly carried out, complete and permanent success may usually be expected even in the worst cases.

Epilepsy.

Symptoms and Diagnosis.—Ordinary idiopathic epilepsy, in the form of *grand* or *petit mal*, often begins in early childhood. In Fig. 219 is shown the age of onset of the condition

in 81 cases observed in hospital and private practice. It is certain, however, that a large number of the cases in children which look at first like regular epilepsy turn out to be merely symptomatic convulsions. They may sometimes prove to be due to past or present organic brain disease. Often they are merely functional disturbances, set up or predisposed to by pyrexia, intestinal toxæmia, or emotional excitement in children with an impressionable nervous system ; and this may happen in older children as well as in infants.

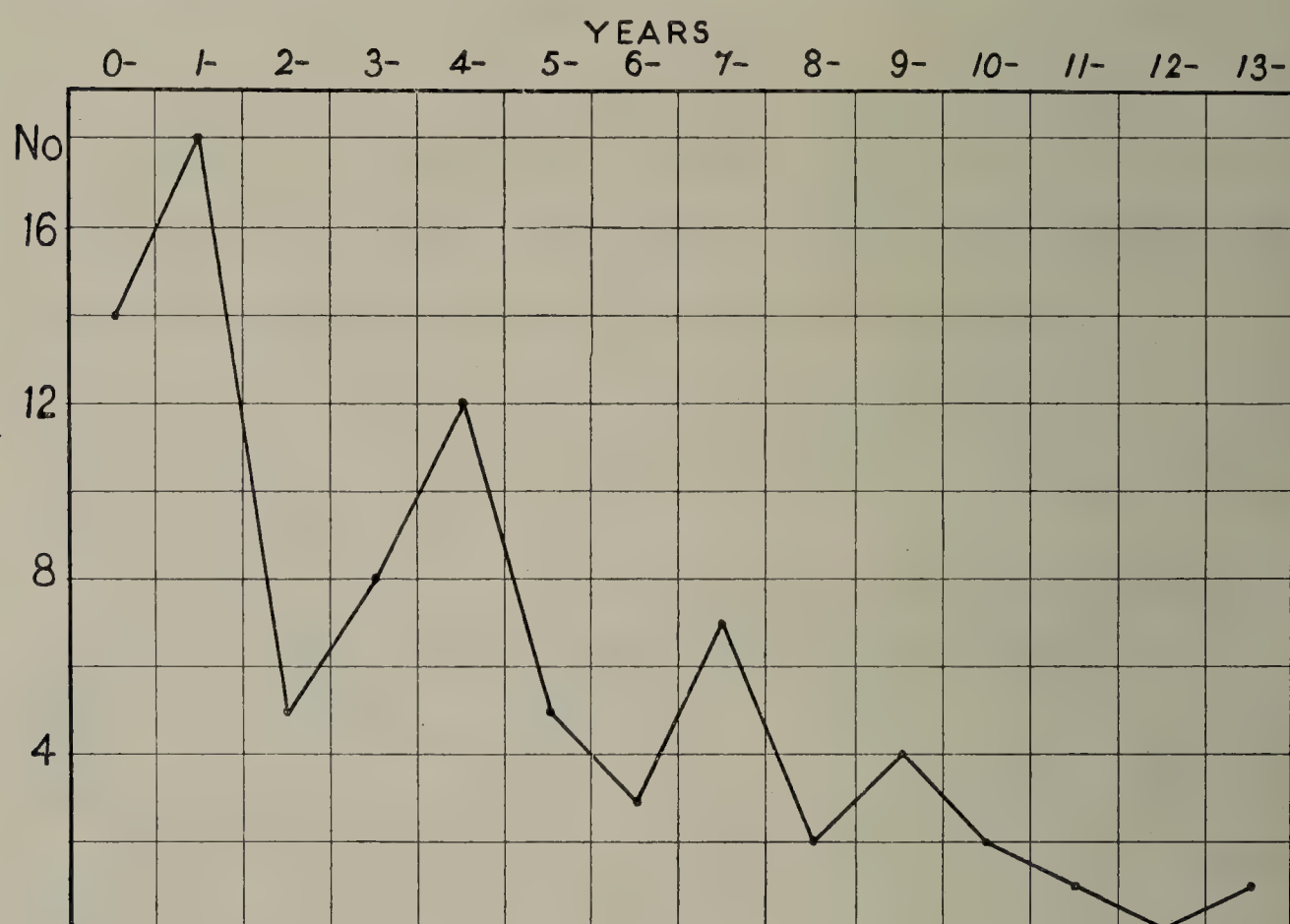


FIG. 219.—Showing age of onset of Fits in 81 cases (48 private and 33 hospital) of Epilepsy.

The question whether, in a given case, the convulsions are truly epileptic or merely symptomatic of a passing disturbance, is of importance with regard to the prognosis ; but the differentiation between the two groups of cases is not easy. It is generally impossible to speak positively as to the nature of the case at the time of the first fit, and our opinion should always be guarded. The following particulars, however, have considerable significance. If fever, acute dyspepsia, or any other indisposition is present—and, in the case of repeated convulsions, if this was also the case on the former occasions—this is strongly in favour of the case being symptomatic. The

occurrence of the seizure during the night or early hours of the morning, and the presence of a family history of epilepsy, are in favour of the case being idiopathic.

A large proportion of the children who have suffered from true epilepsy for any length of time have also more or less noticeable mental peculiarities. Dawson and Conn¹ have shown that the average intelligence of epileptic children is below the normal, that there is a tendency to deterioration with the progress of the disease, and that with a cessation of the fits there may result a definite improvement. In some cases the disease causes a tendency to mental exaltation, an abnormal absence of shyness, a want of mental balance, or an uncertainty of temper. The presence of peculiarities of this kind greatly increases the probability that the case is one of true epilepsy.

In a large proportion of the cases of true epilepsy in childhood the child neither wets himself nor bites his tongue during the attack.

Prognosis.—The prospect of recovery in cases of epilepsy is always a question of very great anxiety. The prognosis is much better in the milder cases and when heroic treatment can be undertaken, although one of us (J. T.) has personally only observed a few instances in which true epilepsy in children has recovered completely, and left no bad result of any kind. When the fits continue to recur for any length of time, however, there is almost always some deterioration of the intellect. A perfectly sane epileptic child is not very often seen—at least among those in whom the disease began in early childhood. In most cases in which the fits continue, the mental and emotional peculiarities increase steadily until some degree of dementia results.

Treatment.—It must be appreciated that the treatment of epilepsy is not simply a matter of prescribing sedatives. The whole dietetic and environmental régime must be inquired into and rendered as little stimulating as possible, if the best results are to be obtained.

In the first place, the child should be removed to some place where there is little excitement and a complete absence of all mental worry and strain, and the routine of the day is regulated. The good effect of environment alone in diminishing the

¹ S. Dawson and J. C. M. Conn, *Arch. Dis. Child.*, 1929, iv., 142.

number of seizures is often most striking when a child is simply admitted to hospital for observation. The noise and hubbub of the streets of a busy city are disturbing factors. Residence in the country or by the seaside exerts a sedative effect. The patient should not, however, be without some form of occupation. Helping in a farm or garden will usually supply a sufficiency of entertainment without producing any undue excitement.

Seldom does a child improve if he continues at school. It is only rational that the brain be given as much rest as possible. In any case, whether sedatives are given or not, there is usually a degree of dullness which precludes the child from benefiting by a continuation of his education. After recovering, the normal alertness often returns and the child will easily make up for the time which has been lost.

It has long been recognised that over-feeding and dietetic indiscretions tend to make matters worse. Similarly, constipation exerts a bad effect and hence the diet should be bland and not over-abundant and the bowels kept freely open. Indeed, it was the beneficial effect of a spare diet, and the fact that it had been shown experimentally that acetone bodies exerted a sedative action, that prompted Wilder¹ to test the use of the ketogenic diet in the treatment of epilepsy (Appendix F). The ketogenic diet is rich in fat and poor in carbohydrate and on that account induces a ketosis, *i.e.*, an acidosis due to the excessive production of ketones (acetone bodies) (p. 553). There never results any symptoms of acidosis from this therapy and the only untoward manifestations are sickness and vomiting due to the nausea induced by the excessive amount of fat in the diet.

Much has been written regarding the efficacy of this form of treatment in epilepsy. Talbot² of Boston, and Helmholz³ of the Mayo Clinic, are its great advocates, but even they admit that it is no panacea, that many cases do not react to it, and that it is always of the nature of an experiment. In our experience this form of therapy, when carried out in hospital, has had no more effect than the regular dietetic and other routine of the hospital ward.

¹ R. M. Wilder, *Mayo Clin. Bullet.*, 1921, iii., 307.

² F. B. Talbot, *Treatment of Epilepsy*, New York, 1930.

³ H. F. Helmholz, *Journ. Amer. Med. Assoc.*, 1927, lxxxviii., 2028.

As a rule, for a time at least, some sedative must also be employed. Luminal and some form of bromide are those usually prescribed. Since luminal is the least depressing, and has little effect on digestion, it is wise to try it in the first instance. It can be given in half to one or more grain doses twice or thrice daily. If luminal has no marked effect then resort can be had to one of the bromide preparations—ammonium, sodium, or potassium. Of these three preparations, ammonium bromide is the least depressing and is to be preferred in the case of children. Two to four grains thrice daily of any one of the salts of bromine may be given safely to a child of twelve months. For older children larger doses may be required: the drug can be pushed till it exerts its physiological effect. The great disadvantage of these preparations is that they upset the appetite and are constipating, so that special attention must be paid to the action of the bowels. Another drawback to these preparations is that some patients possess an idiosyncrasy and develop a rash. The addition of a small amount of arsenic—*liquor arsenicalis* i minim—to each dose, however, lessens the tendency to a bromide eruption (p. 397).

Pyknolepsy.

The term “Pyknolepsy” has been applied to a kind of slight recurrent epileptiform attack occurring in childhood which differs essentially from true epilepsy in being generally recovered from entirely, and in being not affected at all by the remedies which usually benefit cases of that disease. The condition was first described in 1906 by M. Friedmann¹; and, as his observations have been confirmed by many subsequent writers,² it seems to be one of real importance in practice.

The attacks usually set in suddenly in healthy children between four and twelve years old, and consist in a brief, though not entirely complete, loss of consciousness. The limbs suddenly relax, though the child does not usually fall, or drop his toys, or change colour; the eyes stare and turn up, and there is a

¹ “Ueber die nicht epileptischen Absencen oder kurzen narkoleptischen Anfälle,” *Deutsche Zeitschr. f. Nervenheilk.*, 1906, xxx., 462; *Zeitschr. f. d. ges. Neurol. u. Psychiatrie*, 1912, ix., 245.

² W. J. Adie, *Proc. Roy. Soc. Med.* (Neurol. Section), Mar. 1924, xvii., No. 5, p. 49.

slight flicker of the eyelids. After some five to ten seconds he comes rapidly to himself, and is apparently little if at all put about by the attack. He evidently feels quite well after it, and goes on cheerfully with his play as if nothing out of the way had happened.

These small fits are numerous from the first—from 6 to 100 in the day—and their characters are always the same. Sedatives such as bromides and luminal have no effect on them.

They cannot at first be distinguished for certain from *petit mal*; but, as the case goes on, their *frequency*, the *unvarying mildness and uniformity of their symptoms*, the *absence of any bad effect from them on the child*, and their *resistance to all the usual sedatives*, may be held to complete the diagnosis.

No treatment seems to have any effect on their character or recurrence, but after a time, which varies in different cases from a few weeks to nine years—the average duration being three years—they spontaneously and entirely cease. Nothing is known of their etiology.

Neurasthenia.

Neurasthenia may be defined as “hyper-sensitiveness of higher centres with proneness to general exhaustion of nervous energy, mental and physical; whilst hysteria is characterised by excitability and explosiveness of the higher centres, associated with local sensory and motor paralysis, and deficient powers of control of emotional display.”¹ The two conditions are often met with together.

One most typically neurasthenic child was a bright pleasant-looking little girl of nine, who came of a very neurotic family. Her general health had been good; but all her life she had been subject to symptoms of mental and bodily exhaustion after excitement. So long as she lived quietly with adult relatives who were sensible people, she was like any other happy, contented child—obliging, good-natured, and thoughtful for others, fond of animals and very patient with them. She was keenly interested in most of her lessons and was very well read for her age. She had never been regularly at school for a long period, for, after repeated attempts, it had been found that

¹ Leonard Guthrie, *Functional Nervous Disorders in Childhood*, London, 1907, 15.

she was quite unable to bear the strain of ordinary school attendance. When sent to school she would be full of interest in her lessons for the first few days; and her teachers, being much impressed by her intelligence and originality, would predict great progress. In a short time, however, she would become tired and irritable, and in spite of being given less work and allowed to take a day in bed occasionally, she would get so spiritless and muddled that her schooling had to be stopped altogether. Once, when seven years old, she was severely frightened by a tramp who tried to assault her. Just before, she had been making remarkable progress in arithmetic. After the fright, she seemed collapsed, and at school was found to have lost for the time all power of doing even the simplest sums.

In cases of this kind the main treatment consists in attention to the bodily health, in avoiding all forms of excitement at home, and especially in strictly limiting the extent to which the patient associates with other children in school or elsewhere, so long as doing so is found to be exciting and injurious.

Hysteria.

Manifestations of hysteria are rare in children under six years, but occasionally they are met with even in infants under two. As has been already pointed out (p. 701), the place occupied by hysteria in later life seems to be taken to some extent in infancy by various of the so-called "bad habits."

In children between nine years and puberty who belong to neurotic families, hysterical affections are not uncommon; and boys are often affected, though not so frequently as girls. In these older children, also, hysterical symptoms are apt to be associated with chorea, habit spasm, epilepsy, and various forms of mental disease.

Symptoms.—Hysteria may manifest itself in childhood, as in later life, either by motor, sensory, or psychical symptoms. These are usually less complicated than in adults. Among the sensory disturbances, hyperæsthesia or pain on movement of some part is common. Headaches are also often complained of. Anæsthesia is only rarely found. Hysterical joint affections have been already dealt with (p. 86). Motor symptoms, such as contractures of the limbs or, less commonly, paralysis,

are frequently seen, and the muscles of the larynx are among those sometimes implicated. Painless and effortless vomiting after meals is not very rare in older girls, and it must be remembered that something like it sometimes occurs in babies.

An interesting motor symptom which is occasionally met with in hysterical babies, and which is sometimes mistaken for spastic diplegia, is the so-called "*flexibilitas cerea*." In this the child's limbs are held stiff; but can readily be put into any position and remain so for a long while, the child looking



FIG. 220.—*Flexibilitas Cerea*. (Girl of 17 months.)

absorbed and gratified all the time (Fig. 220). The symptom usually begins after some weakening ailment such as an attack of diarrhoea. If much sympathetic attention is given, it will continue indefinitely and become more marked. Even under sensible treatment it may last for months. The treatment, apart from attention to the general health, consists in distracting the child's mind from his symptoms and getting him interested in outside things; and especially in inducing him to use his limbs freely.

The mental condition in hysterical children is generally characteristic. They show the usual intense interest in their symptoms and a distinct gratification in having them examined and talked about. In our experience the intelligence of

hysterical patients is usually below the average.¹ In older girls we sometimes meet with mental depression, accompanied by anorexia and obstinate constipation.

The **diagnosis** of hysteria is made mainly, as in adults, by excluding the presence of organic disease sufficient to cause the symptoms. We must, of course, always bear in mind the *possibility* of organic disease and hysteria being co-existent. The patient's demeanour is often quite characteristic; if a girl tells you with a placid smile that her pains are "agonising," you may know that they are certainly hysterical in nature.

The **treatment** is generally most satisfactory, provided the child can be removed at once to a hospital or nursing home, or to the house of a judicious friend. Such a change of surroundings, with the elimination of morbid sympathy, is often all that is necessary for a rapid cure. Sometimes, however, if the environment is not satisfactory, much patience as well as tact may be required.

When the child is emaciated, and has loss of appetite and constipation, a course of general massage and feeding-up (Weir-Mitchell) is of great value. If the limbs are affected, local massage, faradism, and douching are indicated. Laxative and tonic medicines have a useful function, and sometimes valerian seems to do good. During such bodily treatment it is of the greatest importance that every effort should be made to make the child happy and hopeful, and to transfer her interest from herself to other things. Bad habits should be watched for and checked if present. Cases of hysterical vomiting are sometimes benefited by drop doses of liquor arsenicalis, or two drops of laudanum immediately before food. Babies with neurotic vomiting are also sometimes benefited by small doses of liquor arsenicalis ($\frac{1}{4}$ to $\frac{1}{2}$ a drop).

When the patient has begun to improve satisfactorily, she will benefit from a thorough change of scene, and nothing is better for her than the varied open-air interests of farm life. When her health seems re-established, a happy, well-regulated school routine will be useful in promoting healthiness of mind and preventing a recurrence of the hysterical symptoms.

¹ S. Dawson, *Intelligence and Disease*, Med. Res. Council Spec. Report, No. 162, 1931, p. 25.

Habit - Spasm (*Habit - Chorea*).

Habit-spasm is a fairly common affection, both in boys and girls, between six years old and puberty. It often appears in those whose bodily strength has been lowered by illness or bad feeding, or who are suffering from nervousness either from mental strain, worry, or shock of some kind, and sometimes in children who have been spoiled.

Symptoms.—The twitching movements which are characteristic of the disease are most frequently seen on the face or head. There may be blinking of the eyes, screwing up of the nose, a sudden nod or shake of the head, or a shrug of the shoulders. Sometimes there are grasping and other movements of the hands, or jerking of the lower limbs and trunk. Frequently, in the severer cases, there is a tendency to repeated noisy clearing of the throat, or to the sudden ejaculation of some word; and the word may be a specially undesirable one (coprolalia).

Diagnosis.—In typical cases of habit-spasm the movements differ considerably from those of ordinary chorea, being quicker and less extensive, and also being repeated over and over again in the same way, instead of being varied irregularly. Another point of difference is the effect which the presence of onlookers has on the movements. In habit-spasm they are often diminished or stopped when the child knows he is being watched, while the movements of ordinary chorea are more apt to be exaggerated under these circumstances. Atypical cases of habit-spasm may, however, at first sight look very like chorea. Besides the characteristic movements, the children may show excitability, irritability, passionateness, and obstinacy; and sometimes are difficult to control. Many sleep badly or have headaches. Unlike chorea, habit-spasm is apt to spread by imitation; and, also unlike chorea, it may last for years. Although it is not a manifestation of rheumatism in the sense that chorea is, it is certainly not uncommon to see it in rheumatic children.

The **treatment** of this apparently trivial complaint is often extremely difficult and disappointing; and, as Still says, "there are few disorders of childhood out of which the medical attendant is likely to gain less credit." The fact of the disease occurring at all shows that the child's nervous system is in an unsatisfactory state; and in all well-marked cases, before we

can hope for recovery, a complete and prolonged change in his surroundings and in his ways of life is called for. A rapid cure can never be expected. The main indications for treatment may be stated as follows:—

1. The child's mental condition must be cared for. All sources of distress, such as fear of punishments or bullying must be stopped. He must receive every encouragement, and be kept fully occupied in a pleasant way.

2. If the movements are severe, the patient may be urged to use his self-control to check them, but punishment or even scolding is injudicious. Constant fault-finding is also bad, because it keeps the child's attention fixed on the habit.

3. School attendance must usually be stopped; but it is often good for the child to have an hour or two of lessons in the day. The subjects chosen should, at least at first, be those to which he has least objection, and those over which he does not become excited.

4. The child should be encouraged to sleep as long as possible, and all causes of excitement must be avoided.

5. He must, if possible, be made fat; and, if there is distinct malnutrition, it is sometimes a good plan to begin with a course of Weir-Mitchell treatment. Generally, however, moderate exercise, feeding-up with extra milk, congenial companionship, and a happy out-of-door life are what is wanted.

6. Any local sources of irritation, such as adenoids or refractive errors, should certainly be attended to.

7. In most cases drugs are of little importance. If the child is anæmic, however, he will probably benefit greatly from a course of iron. A combination of arsenic and bromide of potash is recommended by some. Still has seen benefit from ergot and nux vomica (ext. ergot. liq., half to one drachm; tinct. nuc. vom., 5 minims t.i.d.). Valerian seems also, at times, to have a good effect. Electricity has been much used, and is thought by some to do good.

Spasmus Nutans (*Head-Shaking with Nystagmus*).

Spasmus nutans is a functional co-ordination neurosis affecting infants under two years, and is seen mostly in poor children who live in dark rooms. It generally begins between the fourth and twelfth months—at an age when the baby is

spending much of his time and energy in perfecting himself in the difficult art of raising his head, turning it round, converging his eyes, and focusing his accommodation.

Symptoms.—The two principal symptoms—involuntary nodding or shaking of the head, and ocular nystagmus—are usually both present, but either may begin some weeks before the other. Sometimes head movements alone are observed; and in other cases only the peculiar nystagmus is seen. The movements of the head may consist in simple forward nodding, but lateral or rotatory shaking is commoner. They cease when the child is lying down, and also when the eyes are closed,



FIG. 221.—Attitude in Spasmus Nutans. (Girl of 20 months, showing peculiar way of holding the head back.)

voluntarily or otherwise. The nystagmus is rapid and of short range. Its unique character has already been described (p. 677); and it is so distinctive that it is easily recognised, even when unaccompanied by head movements. The nystagmus is generally increased in extent when the head is held steady, and sometimes it only begins when this is done, or when the child looks to one or other side.

Rhythmical contraction and dilatation of the pupils (hippus) may sometimes be found; and, occasionally, convergent strabismus develops. The child has often a peculiar trick of turning his head to one side and staring fixedly out of the opposite corners of his eyes—usually in an upward direction. This gives him a curious preoccupied look. In other cases he throws back his head and looks with his eyes directed downwards as if he had bilateral ptosis (Fig. 221).

The exact explanation of this symptom is obscure, but there seems no reason whatever to regard it, as some have done, as a sort of *petit mal* attack. The intellect is never affected as a result of spasmus nutans. It should, however, be mentioned that children whose intelligence is below the average seem rather apt to be affected with the disease. The movements of the head are, to a large extent, beyond the child's control. They are noticeably increased when his attention is aroused. For this reason they are generally at their height when the child has come through crowded streets to the waiting-room. They diminish with drowsiness, and cease during sleep.

The symptoms usually begin suddenly, and almost always in mid-winter. They seldom last less than six weeks, usually three to six months, and sometimes longer. The condition always ends in complete recovery, with the exception that, should strabismus occur, it may be permanent. Occasionally the symptoms recur in the following winter, and we have once seen them do so in a third year.

Causation.—The causation of this disease, like that of chorea, is somewhat complicated.¹ Among the chief factors we may reckon the child's age, the absence of sufficient sunlight in his surroundings, and the presence of rickets. Falls on the head and the irritation of teething seem sometimes to act as determining causes, and anything that lowers the general vitality may probably be regarded as predisposing. We have never found any connection between this ailment and ear disease.

One of us (J. T.) has seen one instance in which a father and daughter both suffered from spasmus nutans and nystagmus, at the ages of 14 and 16 months respectively.

Diagnosis.—The condition is generally easy to recognise. Eclampsia nutans, or the "salaam convulsion," has sometimes been confounded with it. That is, however, quite a different disease, being a form of epileptic seizure met with in mentally defective children and associated with grave cerebral lesions (p. 674).

The deliberate rhythmical jerking or shaking of the head

¹ W. B. Hadden, *Lancet*, 14th, 21st, and 28th June 1890, 1293, 1349, and 1416, and *St Thomas's Hosp. Rep.*, 1890, N.S. xx., 205; R. W. Raudnitz, *Jahrb. f. Kinderheilk.*, 1897, xlv., 144; H. Rietschel, *Charité Annalen*, 1906, xxx., 152; Thomson, *Scot. Med. and Surg. Journ.*, July 1900, vii., 7; and *Brit. Med. Journ.*, 30th Mar. 1901, i., 763.

in neurotic children, referred to earlier in the chapter (p. 705), is also easily distinguished from spasmus nutans. In it the movements have a wider range, nystagmus is absent, and the head-shaking ceases when the child's attention is arrested and increases when he is left to himself. It is also generally seen in rather older children.

Treatment.—Spasmus nutans usually improves steadily and satisfactorily under antirachitic and tonic treatment. The chief thing is to secure abundance of fresh air and sunshine, and to give cod-liver oil. While sedatives such as antipyrine seem, in some cases, to diminish the movements, they cannot be held to be of much importance.

CHAPTER XXIX

SOME FORMS OF PARALYSIS

THE various forms of paralysis met with in children may be conveniently divided into two groups, according as the symptoms have dated from birth or have set in during infancy or later childhood.

Paralysis dating from Birth.

Paralysis which is present from birth may be due to intra-uterine disease or to arrest of development; but it is often the result of an injury during delivery.

Peripheral Birth Palsies.

In peripheral "birth palsies," the nerves of either the face or the arm, or sometimes of both, may be involved; the lower limbs are rarely damaged, because their nerves lie so deeply.

Facial Paralysis occurs in new-born children as the result of injury to the facial nerve during delivery. It is generally due to the pressure of the forceps; but, in a few cases, the promontory of the sacrum may be to blame. The weakness is usually recovered from in a few days or, at most, within a fortnight. Occasionally, however, it lasts for months or may possibly be permanent.

The diagnosis seldom presents any difficulty, as the only condition which may be mistaken for it is *congenital defect of the nucleus of the seventh nerve*.¹ This lesion should always be remembered when birth palsy of the face does not recover. Should both sides of the face be affected, or one or more of the ocular muscles be also paralysed, these are points in favour of this diagnosis.

The treatment of facial paralysis from birth injury is largely

¹ H. M. Thomas, *Journ. of Nervous and Mental Disease*, Aug. 1898, xxv., 571; Heubner, *Charité Annalen*, xxv., 1900, 211; Rainy and Fowler, *Review of Neurol. and Psychiat.*, Mar. 1903, i., 149.

expectant; but, if recovery is delayed, gentle massage may be used.

Paralysis of the Arm arising during delivery is due to injury of the cords of the brachial plexus. It may occur



FIG. 222.—Erb's Paralysis of both Arms. (Girl of 3 months.) Breech presentation. (Dr Peter Davidson's case.)

in head presentations, but is commoner in breech cases. It may be caused by direct pressure of the end of the forceps, or by the accoucheur's finger, provided the force is applied while the plexus is on the stretch owing to the head and the shoulders being pulled in different directions. Sometimes,

apparently, it results from severe traction on the arm only, or from lateral twisting of the head during the extraction of the child.¹ Occasionally both arms are affected (Fig. 222).

In the great majority of cases it is the upper cords of the plexus (fifth and sixth C.) that are implicated, and the paralysis is then of the "upper-arm type" (*Erb's paralysis*). In these the position of the arm is very characteristic. The shoulder is fixed and the elbow fully extended, while the humerus is rotated inwards and the forearm pronated, so that the palm turns backwards or even outwards. After a few weeks there is flattening of the shoulder and atrophy of the upper arm. The forearm is not wasted, and the hand movements are little interfered with. The muscles involved in a typical case are the deltoid, biceps, coraco-brachialis, and brachialis anticus, the supra- and infra-spinatus, the teres minor, the supinator longus, and sometimes the supinator brevis. No sensory changes are found.

The "lower-arm type" (*Klumpke's paralysis*) is less common. In it the lower cords (eighth C. and first D.) are affected. The paralysis affects the small muscles of the hands and the flexors of the fingers in the forearm, and there is some anæsthesia in the ulnar region. There may also be myosis on the side of the lesion, with sluggish contraction of the pupil and diminution of the palpebral fissure.

Cases of both types are apt to vary considerably in the exact distribution of the paralysis, owing to irregular involvement of the cords. Sometimes there is separation of the upper epiphysis of the humerus, or fracture of the clavicle or humerus, in addition to the nerve injury. Dislocation of the shoulder-joint may also occur; and in many cases a secondary subluxation of the head of the humerus takes place and constitutes a troublesome complication in the treatment.²

The **diagnosis** is easy, especially if it is known that the paralysis has existed from birth, and that the birth was a difficult one. Should no clinical history be forthcoming, however, the case may be mistaken for one of poliomyelitis.

Treatment.—As soon as possible after recognition of the paralysis the arm is put in the position in which the affected

¹ E. F. Trevelyan, "Peripheral Birth Palsy," *Quart. Journ. Med.*, 1908, ii., 1908-9, 405.

² Whitman, *Orthopædic Surgery*, 6th edit., London, 1919, 469.

muscles are completely relaxed. The arm is abducted through 90° , flexed at the elbow, and rotated outwards, so that the forearm is pointing upwards and supinated with the palm

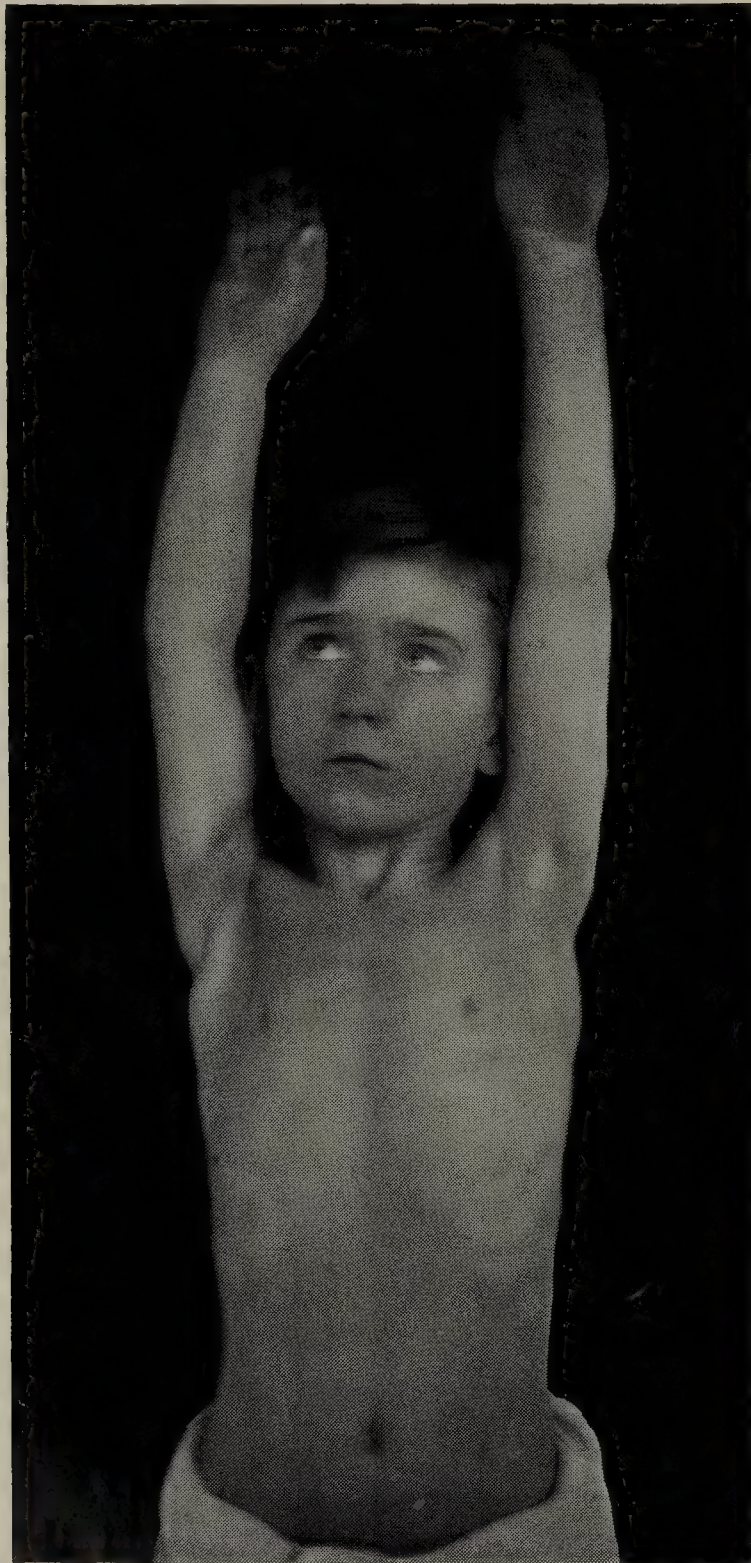


FIG. 223.—Boy aged $6\frac{1}{2}$ years who had made a good functional recovery from a birth paralysis (Erb's type); note, however, marked shortening of the affected (right) arm.

facing the side of the head. The arm may be fixed in this position by plaster of Paris or other method, but the best is a splint made of pexuloid or vulcanised fibre, as this is light in weight and can be removed easily for purposes of cleaning. The splint consists of a wide body-piece moulded to the chest with a right-angled piece springing from the upper and axillary border to support the arm. This splint must be worn night and day for at least six months, unless complete recovery takes place earlier. The arm is freed every day and very gently manipulated to avoid fixation of the joints. As recovery takes place, re-education of the movements may be tried by encouraging the child to catch an object held in different positions, all the time the arm being supported

to guard against undue stretching of the weakened muscles. Very gentle massage may be of some assistance, but electricity is of no value.

In many cases complete or almost complete recovery of

power takes place within two or three months. In some, however, the improvement proceeds more slowly, and yet the child entirely recovers. In the majority of instances, although no actual paralysis is left, the deltoid and supra- and infra-spinati remain weak with some shortening of the arm (Fig. 223).

When no improvement is noticed within three months, especially in cases of the lower-arm type, a surgical operation may be recommended to remove the cicatrised portion of the injured nerves and bring the uninjured parts together. The advisability of such an operation is a difficult question in view of the surprising amount of spontaneous improvement which frequently occurs even at a late date, and because, in severe and long-standing cases in which no improvement has occurred, the damage present is often so extensive that the uninjured parts of the nerves are too far separated from one another for it to be possible to bring them together.

Cerebral Paralysis and other Cases of Brain Lesion due to Injury during Labour.

We meet, now and then, with cases of spastic diplegia or other cerebral paralyzes dating from birth, in which there is no reason to suppose that any birth injury has occurred to account for the condition. In these the lesion causing the paralysis may be some developmental abnormality of the central nervous system, or possibly an intra-uterine brain disease or injury. Collier¹ believes that *agenesia* of the pyramidal tract is a common cause of spastic diplegia, but in view of the large proportion of difficult labours, 50 per cent. at least, met with in these cases, injury at birth would seem to be a not unimportant etiological factor.

Cerebral Birth Palsies (*Spastic Diplegia—Little's Disease*).

Causation.—The reasons for regarding these cases as mostly of traumatic origin may be given as follows. When we make careful inquiries from mothers, and from other sources, regarding a large number of young children with spastic diplegia, we cannot fail to be struck by the very large number of histories we get of severe or abnormal labours; while such histories are comparatively rare in cases in which the children are healthy.

¹ J. Collier, *Brain*, 1924, xlvii., 1.

This experience suggests that an injury at birth is a common cause of this condition, and this view is strongly corroborated by pathological evidence.

Herbert Spencer,¹ F. J. Browne,² Eardley Holland,³ Ehrenfest,⁴ and others, have shown how very often an infant that dies during or soon after labour is found to have had an intracranial hæmorrhage; and that such injuries often occur under conditions which are quite common in the course of obstetric practice.

It has also been demonstrated by post-mortem examinations that many infants who have died within a few weeks of a difficult birth, and who, while they lived, had shown the usual symptoms of spastic diplegia, show intracranial lesions precisely similar to those found in the still-born babies, though sometimes less extensive.

There is no doubt that a large proportion of cases of spastic diplegia are the result of birth injuries. These injuries seem usually to arise from undue pressure on the head caused by the use of forceps, or other active measures employed to complete the delivery; and sometimes they are due, in prolonged labours, to the strong but unsuccessful attempts of Nature to deliver the child spontaneously. The special engorgement of the cerebral circulation usually present during difficult labours acts strongly as a predisposing factor and favours the tendency to hæmorrhage. Hæmorrhage is also particularly apt to happen when the child is premature, and when the confinement has been at all hurried.

The diagnosis, in any case, that the head has been injured is strongly confirmed if, in addition to the abnormal character of the labour, we find that the baby showed at birth external evidence of force having been employed, and especially if he has had symptoms of an obviously cerebral nature. The external injuries may be of various kinds. There may, for example, have been severe bruising or laceration of the scalp,

¹ "On Visceral Hæmorrhages in Still-born Children," *Trans. Obstet. Soc. London*, 1891, xxxiii., 203.

² "Still-birth: its Causes, Pathology and Prevention," *Edin. Med. Journ.*, Sept. 1921, 153.

³ *Report on the Causation of Fœtal Death*—(Ministry of Health), London, 1922.

⁴ *Brain Injuries of the Child* (Appleton), New York, 1922.

a cephalhæmatoma, or paralysis of the face or of an arm; in other cases, the clavicle or humerus has been broken, or a sternomastoid tumour has formed. Occasionally we may hear from the obstetrician that the fontanelle was much distended at birth, or that the cerebro-spinal fluid was found on lumbar puncture to be blood-stained.

Of a series of 37 cases observed at R.H.S.C., Glasgow, in only 15 was the labour described as normal; 15 were instrumental or difficult births; in 3 there was difficulty in getting the child to breathe; 2 of the infants had fits during the first week of life and in 2 cases the birth was precipitate. A percentage of 60 abnormal deliveries is high and must have some significance. Of a series of 100 examples of pneumonia, also observed in R.H.S.C., Glasgow, in 92 the labour was said to be normal: in 7 the delivery was instrumental and in one a difficult breech case.

It has been suggested that syphilis may play a part. This has seldom been the case in our experience. Of 27 examples submitted to Wassermann's test, only 2 reacted positively.

Symptoms.—In many cases the infant, when born, is more or less collapsed or asphyxiated, so that vigorous and prolonged attempts at artificial respiration are found necessary to induce him to breathe. Often he has one or more convulsions or severe twitchings on the first day or later, and sometimes these continue to recur during the early months or even longer. Sometimes, during the early days of life, there is constant screaming and sleeplessness or the child may be unnaturally drowsy.

The most striking characteristic of these cases in infancy, apart from the early symptoms just mentioned, is the *spastic rigidity* of all the muscles. The distribution of this varies in different cases according to the position and extent of the lesion. The lower limbs are usually more severely affected than the upper, and sometimes they only are affected. Occasionally it is limited to one side of the body. In the large majority of cases, however, all the four limbs are more or less implicated; and it is very characteristic of those with a traumatic origin that the spasticity is distinctly greater on one side than on the other.

In severe cases (Figs. 224 and 225) the whole body is rigid. The back and hip-joints are extended, so that the child cannot

sit; the adductors of the thighs are so spastic that the legs are firmly crossed whenever the child makes any attempt to bring one of them forward; the feet are pointed and the toes dorsiflexed. This constant overaction of the muscles, being of a purely tonic nature, does not, however, cause them to hypertrophy. The knee-jerks may be greatly increased but, in severe cases, they are generally only obtained with difficulty,



FIG. 224.—Spastic Diplegia with mental defect from cerebral hæmorrhage at birth. (Girl of $7\frac{1}{2}$ years.)



FIG. 225.—Same condition in a girl of $8\frac{1}{2}$ years, with considerable intelligence.

because the general rigidity prevents all sudden movements. Well-marked ankle clonus is rare. The plantar reflexes show a distinct extensor response.

Although the upper limbs are usually less affected than the lower, the child generally has difficulty in grasping objects, and when grasped, in letting them go again.

As they grow older a number of the children develop an intention tremor or athetoid movements of the limbs.

Although the rigidity of the limbs is obvious when we look

for it, it is sometimes overlooked for a long time by the parents ; and they only become concerned about it when they find the child late in being able to balance himself in sitting and standing. Their anxiety may, however, be aroused by the way he often over-extends his spine and throws his head back when he is excited.

The facial muscles are also not under proper control, and they tend to overact when he tries to speak, so that he grimaces in an exaggerated way. Speech is late in being acquired, and is guttural and badly articulated.

In some cases the cranium is normal in size and shape ; but, in most, the head remains too small, owing to the growth of the damaged brain not keeping pace with that of the body. When this is so, the fontanelle may close too soon—between the sixth and tenth months—though never quite so early as it often does in microcephalus. The head may also be obviously deficient in the frontal or occipital regions, or too high in the vault. In some cases there is enlargement from hydrocephalus ; in others there is a vertical ridge down the centre of the forehead.

Although severe spasticity of the limbs is such a prominent symptom in typical cases of spastic diplegia and so important in their treatment, there are many in which it is very slight in degree, so that it forms a much less striking feature in the clinical picture. The mental defect in these less spastic cases is often not very severe, but sometimes it is as bad as in the others.

The other main symptom present in most cases of spastic diplegia, is *an arrest of mental development*. This is of course much longer in being noticed than the muscular rigidity. Among the young children brought to the Edinburgh Children's Hospital on account of mental defect, a considerable proportion belong to this type. Out of 950 such children, under five years, 134 (*i.e.* 14 per cent.) had spastic diplegia. In 82 of them, the nature of the labour, the child's state at birth, and the other conditions present, led one to conclude that there had certainly been a severe birth injury ; and in 18 others there was some reason to suspect this. In the remaining 34 there were no grounds for such a diagnosis, although in a few of them a latent injury may have existed.

The mental condition in different cases varies greatly

according to the degree and position of the lesion. In many it is profoundly affected, in others slightly, and in a few (15 out of 149) not at all. On occasion the children have an intelligence above the normal. Dawson and Conn,¹ working in R.H.S.C., Glasgow, found the average mental ratio of eight cases to be 88.25; the highest was 119 and the lowest 73. Speaking generally, however, it may be said that in spastic diplegia the intelligence is usually much greater than the vacant expression and facial contortions would lead us to expect.

Prognosis.—In trying to forecast the future in these cases, we have to be guided by the degree of the spasticity, the growth of the cranium, whether there are recurrent convulsions, how far the mental powers seem to be affected, and especially by the extent to which they have already improved. In the early cases, however, we must also remember when forming our prognosis that there is another thing which keeps these children back besides the structural damage to their brains. This is the way in which the uselessness of their stiff limbs prevents their gaining experience and interest in life in the way other babies do. As Dr H. C. Cameron² has recently reminded us, the very imperfect control they have over their bodies is a serious handicap to their intellectual development, for they miss altogether the useful stimulus to thinking which comes from crawling all over the place and feeling, handling, and moving all sorts of things within their reach. The want of this experience is bound to keep them back, so that they seem to be less intelligent than they afterwards turn out to be. We must therefore make allowance for this temporary element in their dullness and not underrate their chances of ultimate improvement under education.

With regard to the prospect of the child's improvement in the power of using his limbs, we must, however, remember that he will *all his life* be seriously handicapped by the spastic state of his muscles, however good his mental condition may be. Even when the stiffness of his limbs is only slight in degree, it will continue to make it harder for him than it is for other

¹ S. Dawson and J. C. M. Conn, "Intelligence and Disease," *M.R.C. Report*, No. 162, 1931, 25.

² "Late Results of Meningeal Hæmorrhage in the New-born," *Brit. Med. Journ.*, 3rd Mar. 1923, i., 363; and "Intracranial Birth Injury," *Lancet*, 15th Dec. 1923, ii., 1292.

people to do anything with them. He will always, therefore, need a more than average strength of will and perseverance; and this can, of course, only be looked for if the higher parts of his brain have escaped injury. When this is so, however, it is often surprising to what an extent a diplegic youth, who has been well trained during childhood, may succeed in various forms of hand work as well as in ordinary lessons; for, in many cases, behind the ungainly demeanour and grimacing there is a great deal of sound sense and educable intelligence.

Treatment.—The extent to which treatment and training are worth while in these cases depends altogether on the amount of sense and character the child possesses. If his intellect or will-power is severely defective, very little can be done that is worth doing. If, however, as not very rarely happens, he shows increasing sense and perseverance as he grows older, so that he is able more and more to co-operate actively in his own treatment, much may be accomplished, in time, which will make the greatest difference to his future.

Many operative procedures have been suggested to overcome the spasm of the muscles and facilitate walking. Avulsion of the obturator nerves, section of the posterior roots of the spinal nerves (Forster's operation), and excision of the sympathetic ganglia, as proposed by Hunter, have all been tried, but in our experience are not only of no help but decidedly injurious and usually leave the child more helpless than he was before the operation. Simple tenotomy of the tendons of the spastic adductors of the thighs, flexors of the knee and of the tendo Achillis is the most useful proceeding.

The details of such treatment and training are considered in Chapter XXXII., p. 819.

Cerebral Birth Injury without Paralysis.

Among 146 cases of young children with mental defect in which there were good reasons for believing that a cerebral injury had occurred at birth, 91 (62·3 per cent.) had symptoms of spastic diplegia, and 55 (37·7 per cent.) showed no bodily change except debility.

In these *non-paralytic cases* the nervous symptoms during the early days resembled those in the paralytic ones. The children were very helpless at first, owing apparently to the feebleness and inco-ordination of their flaccid muscles. In the worst of

them there was, for long, a complete inability to hold up or steady the head, which rolled about helplessly and fell forward on the chest when the baby was raised. The child was also very long in being able to sit up even with support, or to use his hands. In cases in which the mental condition was not of a low grade, however, the muscular movement usually recovered completely in time. A convergent squint was very common in these children and many of them had recurrent fits, which were often of the *petit mal* type.

Paralysis due to Injury of the Cord.

Crothers and Putman¹ have recently drawn attention to the fact that this type of injury is not so rare as was at one time supposed. The symptoms depend on the level of the cord which is injured, and are characterised by a combination of upper and lower motor neurone lesions, and in this way can be differentiated from peripheral lesions. These authors have shown that this type of injury is most liable to occur in breech presentations when too strong traction is made on the body to deliver the after-coming head.

Prophylaxis of Cerebral Birth Injury.—One of the most important results of the Pre-Maternity Clinics, which we owe to the lifework of the late Dr J. W. Ballantyne, has been to show that cases of cerebral birth injury, due to wrong presentations or wrong positions of the child at the beginning of labour, are preventable in the vast majority of cases. It is now taught in all medical schools that the attending physician must see the mother at frequent intervals during her pregnancy, so as to be able to assure himself as to the position of the child, as well as regarding the measurements of the pelvis and the general state of the mother's health. When this is done, he is able to rectify any abnormal presentation or position, and to form an opinion as to whether the conditions present are such as to allow of a safe normal full-time delivery, or if it will be necessary to induce labour before the usual time, or to resort to Cæsarean section.

Were these precautionary measures universally adopted, it is believed that difficult forceps and breech cases would cease to occur. When that time comes, the number of paralysed and mentally defective children will certainly be greatly reduced.

¹ B. Crothers and M. C. Putman, *Medicine*, 1927, vi., 41.

Paralyses Setting in during Infancy and Childhood.

Paralysis arising in childhood may be due to lesions of the nerves, of the spinal cord, or of the brain.

Peripheral Paralyses.

Facial Paralysis.—In Scotland, far the commonest cause of facial palsy in infants and young children is destruction of the facial nerve, due to tuberculous otitis which has spread into the Fallopian canal. In places where bovine tuberculosis is less prevalent, this form of paralysis is not nearly so common.

Its onset is an urgent indication for surgical treatment of the ear and the outlook as to recovery of the facial palsy is bad, for the destruction of the nerve is usually so complete that we cannot expect any treatment—electrical or other—to do any good. In the course of time, however, if the patient survive, the deformity will be materially lessened by the gradual contraction of the paralysed muscles.

In rare instances, ordinary acute otitis media may cause facial palsy from pressure on the nerve. In these cases the paralysis recovers as the inflammation subsides.

In older children we sometimes meet with ordinary *Bell's paralysis* from exposure to cold. This usually recovers in a short time. The treatment is the same as in adults.

We must also bear in mind that paralysis of the face may occur in acute poliomyelitis, usually along with paralysis elsewhere, but occasionally it is the only lesion. It generally ends in complete recovery.

Facial paralysis of the peripheral type is also met with as a symptom of intracranial disease, especially of tuberculous meningitis and cerebral tumour.

Peripheral Neuritis.—Peripheral neuritis is found most frequently as a sequel to diphtheria (p. 736). Severe multiple neuritis of the extremities occasionally occurs as a sequel to various infectious diseases such as influenza, measles, whooping-cough, and enteric fever. It is usually symmetrical in its distribution, affecting both arms, both legs, or all four extremities. It is often characterised by a degree of sensory as well as motor paralysis, and there may be tenderness along the course of the affected nerves. There usually is more or less definite reaction of degeneration and loss of the deep reflexes.

The distal portions of the extremities are first and most severely affected, the muscles most paralysed being usually the extensors of the foot and hand; the peroneal and musculo-spiral nerves are especially liable to be affected.

The treatment consists in keeping the parts warm, and improving the general strength by careful feeding and tonics, especially cod-liver oil; and later in massage and exercises. If pain is severe in the early stage, local heat should be applied and sedatives given.

In many cases complete recovery takes place, but in some the limb is crippled by permanent contractures (Fig. 226).



FIG. 226.—Deformity from Peripheral Neuritis, which followed measles in a boy of 6 years. Fifteen years later, only slight improvement had taken place.

Peripheral neuritis is also met with as a symptom of poisoning by arsenic and lead.

A polyneuritis is considered by some authorities to be the underlying cause of "Pink Disease" (p. 393).

Lead Palsy.—Paralysis from lead poisoning is not common in children in this country, except in the neighbourhood of lead mines or of manufactories in which lead is much used. Its manifestations must, however, always be remembered in examining obscure nervous cases; because its usual sources are such as to be easily overlooked, unless the nature of the symptoms makes us look specially for them.

Hereditary Cases.—In all districts in which lead poisoning is prevalent, it is well known that married women who are poisoned are liable to be sterile, or to have frequent miscarriages,

and that many of their infants die early in convulsions; also that such things happen oftener when both parents are affected. It is perhaps less generally recognised that the infants of healthy women whose husbands are suffering from plumbism may, though less frequently, be affected in a similar way (p. 675).

Sources of Poisoning.—Water which has remained long in leaden cisterns or pipes is one of the main sources. Breast-babies may suck the lead from their mother's nipples if she is using acetate of lead ointment or lotion, or a lead-containing cosmetic. Bottle-fed infants sometimes get it from rubber nipples which contain lead. Chrome yellow is another possible source. It may be sucked from a paint-brush or taken as colouring matter in cakes or sweets. Holt records a number of fatal cases due to children nibbling lead-containing paints from their bedsteads or from other furniture; and Lockhart Gibson has seen a large number of non-fatal cases from white-lead paint.

In 1897 Dr J. Lockhart Gibson of Brisbane drew attention to the fact that the *optic neuritis and ocular paralysis*, which were met with in a large number of Queensland children, were due to acute lead poisoning.¹ Some years later he succeeded, in a series of interesting and convincing papers,² in showing how it came about. The children were in the habit of spending much of their time in verandahs covered with white-lead paint. When this had been exposed to the hot sun for a year or two it became dry and powdery; and the powder, which was largely composed of soluble carbonate of lead, adhered freely to the children's perspiring fingers. It was found that the poisoning only occurred in those who were in the habit of either biting their nails or sucking their fingers; the others escaped. When the paint was freshly applied, it was also apt to stick to the children's hands and feet.

The main symptoms in these cases were paralysis or paresis of one or both external recti, with optic neuritis or choked disc, a blue line on the gums, and the presence of lead in the urine. They set in suddenly with or without colic, vomiting, constipation, and severe headache; and there was often also stiffness with pain in the back and neck. Drop-wrist scarcely ever

¹ *Austral. Med. Gaz.*, 20th Oct. 1897.

² *Ocular Neuritis due to Lead*, Australian Med. Publishing Co., Sydney, N.S.W., 1922.

accompanied the eye symptoms, though it sometimes preceded or followed them. Although this form of lead poisoning does not seem to have occurred extensively elsewhere, Dr Gibson's experience is very interesting and worthy of attention, especially from those who practise in hot climates.

Symptoms of Lead Poisoning.—There is often, although not always, a history of recurring colic and constipation. The characteristic blue line on the gums is said to be present in less than half the cases.¹ The chief peculiarity of lead palsy in childhood is its tendency to affect the lower limbs earlier and more severely than the upper. The usual drop-wrist is, however, seen in some cases, though generally later than the affection of the legs. The excitability of the muscles to galvanism and faradism is much diminished, but there is no reaction of degeneration. The knee-jerks and plantar reflexes are abolished. Paralysis of the ocular muscles, with optic neuritis and occasionally retinal hæmorrhages, is not often seen in this country. There is usually a degree of anæmia and in stained films the red cells show much basophilic or granular degeneration. Lead in small quantity may often be found in the urine.

The *prognosis* should generally be guarded in cases of plumbism. It depends on the duration of the poisoning, the degree of the cachexia and nature of the symptoms. None of Gibson's ocular cases died, though most of them lost their sight more or less completely and permanently. Drop-wrist may be permanent if not treated early. Holt mentions his having seen eight cases of brain affection due to lead, all fatal with one exception.

The *treatment*, after the source of the poisoning has been stopped, consists in encouraging the various excretions, in administering iodide of potash, and in using massage, exercises, and electricity for the paralysed muscles.

Post-Diphtheritic Paralysis.—Diphtheritic palsy not uncommonly follows a slight sore throat which has not been recognised as diphtheritic in origin, as well as more serious cases; its nature is therefore apt to be overlooked.²

¹ G. Variot, "Intoxication Saturnine," *Grancher et Comby, Traité des Maladies de l'Enfance*, t. i., 1008.

² F. E. Batten, "Diphtheritic Paralysis," *Garrod, Batten, and Thursfield's Diseases of Children*, London, 1913, 826.

The *symptoms* may set in within a week of the onset of the throat affection; but generally they do not appear until between the second and fourth, and sometimes not till the sixth or eighth week after the disappearance of the false membrane. The part earliest and most frequently affected is the soft palate; and its implication leads to nasal speech, and often to return of fluids through the nose during drinking. Later, the pharynx and œsophagus may also be paralysed, and the child is then unable to swallow, and coughs and splutters when he tries to do so. Paralysis of accommodation is often added by the end of the third week, so that he is unable to read small print or to thread a needle. In a few cases there is double vision, and ptosis may also occur.

There is often also more or less paresis of the lower limbs, less commonly of the upper, or of the muscles of the back and neck, but the paralysis is never complete. The paretic state of the lower extremities may account for the unsteadiness in, walking which is often present; but sometimes there is also a degree of ataxia of both upper and lower limbs, and, rarely it may be so severe as to suggest the possible presence of cerebellar disease. The knee-jerks are lost early in most cases, though not in all; and they may not be regained for many months. Their early return is a good sign. Sensation is generally little, if at all, affected; but in a few cases there is pain on movement of the legs. Rarely, there is severe and long continued wasting of the muscles.

Paresis of the muscles of respiration is a characteristic and serious manifestation of the disease. It is generally preceded by affection of the palate and is often accompanied by pharyngeal palsy. Paresis of the diaphragm is recognised by observing the respiratory movements of the abdomen. When its action is normal, the downward movement of the diaphragm causes a bulging of the upper part of the abdominal wall with each inspiration; but, when it is weakened by disease, this does not occur, and there may even be an indrawing in this situation. This is, of course, a threatening symptom while it lasts; but fortunately it does not usually last long; and most cases recover, if the accompanying interference with swallowing is treated early by the use of tube-feeding, so as to prevent the occurrence of inhalation pneumonia or bronchitis.

Paralysis of the heart is the manifestation of post-diphtheritic paralysis which gives rise to most anxiety. It shows itself by a feeble and irregular state of the pulse and the disappearance of the first sound at the apex; sometimes also by curious and complicated cardiac murmurs. It sometimes takes place without any other form of paralysis being present; and is not infrequently fatal unless the greatest care is taken. In the worst cases vomiting usually occurs, and this, therefore, is always to be regarded as a very serious symptom.

Diagnosis.—The distribution and gradual onset of post-diphtheritic paralysis are so peculiar that there is rarely any difficulty in recognising the condition. Ocular and palatal paralysis from tumours of the pons and medulla are always accompanied by other indications of intracranial disease. In acute poliomyelitis the loss of power is less general and more complete. Paralysis of the muscles of respiration as an isolated phenomenon scarcely ever occurs except in diphtheria. Some times, indeed, the throat seems healthy and free from diphtheritic organisms; but in these cases it is always possible that one of the other mucous membranes may have been infected.

Prognosis.—Diphtheritic paralysis if untreated by Roux's serum usually lasts for six or eight weeks or a little longer; but it may take many months before the patient is quite well. The weakness of the heart is only slowly recovered from. The disease *always* tends to recover, unless there is heart failure, or serious complications resulting from interference with respiration or swallowing. We have seen one case which made a good recovery from an attack of acute pneumonia; but the crisis took place on the third day.

Treatment.—There is no doubt that Roux's serum, in contrast to Behring's serum, has a marked effect on the course of the disease. This is probably due to the fact that Roux's serum is produced by the injection of young cultures rich in *toxone*, which is the cause of the neuritis, whereas Behring's serum, the usual type employed in this country, is made by the injection of old cultures which are rich in *toxin*. Roux's serum is prepared by the Pasteur Institute, Paris. Injections daily of 10 c.c. for several days is the dosage recommended. Good nursing is specially important in the treatment of post-diphtheritic paralysis. To lessen the risk of heart failure, the

child should be put to bed at once, and kept lying quite flat without even a pillow under his head; and he must not be allowed to sit up *for any purpose whatever*. Food should be given mostly in a liquid form, in small quantities, and at frequent intervals. If the palate is paralysed, the fluids should be thickened by some farinaceous addition; and the child must be fed with great care to avoid choking. Whenever a distinct difficulty in swallowing occurs, recourse must be had at once to tube-feeding (p. 1005). Many children recover when fed in this way who otherwise would certainly have died in a few days. The bowels must be carefully regulated.

The drugs usually given are atropine and strychnine. Batten recommended strongly the subcutaneous injection of $\frac{1}{200}$ gr. of the former and $\frac{1}{120}$ gr. of the latter every four hours. He believed that this has a most valuable effect in warding-off respiratory and circulatory failure. Alcohol should not be given as a matter of routine, but is often useful when there is evidence of cardiac failure. Electricity may do a great deal of harm by exciting the patient.

Spinal Paralysis.

Paralysis of spinal origin occasionally occurs in children as the result of tumour, hæmorrhage, transverse or disseminated myelitis, and, in Pott's disease, from compression myelitis. Various forms of progressive muscular atrophy with a spinal lesion, Friedreich's ataxia, and other rare types of spinal paralysis also develop in childhood. Far the most important disease of the spinal cord that is met with in children, however, is *acute poliomyelitis* (see Chapter XXX).

Paraplegia from Spinal Caries.—In its advanced stages spinal caries may fairly be claimed as a surgical disease. When just beginning, however, before either curvature or suppuration has occurred, it is likely to be seen first by the physician; and he must always be on the lookout for it whenever there is weakness or obscure pains in the lower limbs or pain in the abdomen. When the diagnosis is doubtful, an X-ray examination should *always* be made; and even in obvious cases a plate should if possible be taken before treatment, for future reference.

Paraplegia from spinal disease often begins most insidiously,

and is apt to be overlooked or misinterpreted for a long time. The child, who has generally been falling off for some time previously, begins to show a disinclination for exertion and is more easily tired than he used to be. He often, indeed, denies having any pain; but generally his expression and movements show clearly that he is really suffering considerably. The weakness continues to increase, and at night there may be enuresis, or starting movements of the legs. When he goes about, the child is noticed to hold his back stiffly, and to walk circumspectly as if he were afraid of a sudden jar. At this stage there may be little or nothing else to be made out on examination of the limbs. Usually, however, we find an extensor response, exaggeration of the knee-jerks, and perhaps slight ankle clonus; and, although no curvature may be discoverable as yet, the spinal column is found to have lost the freedom of movement which is characteristic of perfect health in childhood. Later, more pronounced symptoms of spinal caries develop, and some degree of angular curvature generally appears.

In most of the cases of spinal caries in which paraplegia occurs, the disease is situated in the upper dorsal region; for, at this level, the spinal canal is at its narrowest and the cord therefore in special danger of being compressed. When this begins, the lower limbs become spastic and the knee-jerks exaggerated, and the patient may lose control of the bladder and bowel. In older children, a girdle pain is sometimes complained of.

When the lumbar region is the part affected, there is less likely to be deformity, and the knee-jerks may be abolished instead of being increased. If the cervical spine is diseased there is apt to be wasting of the hand muscles as well as spasticity of the lower limbs.

Treatment.—The patient must be kept lying, with or without extension, and his spine rendered immobile by the use of some kind of splint. For this purpose a wooden frame may be used consisting of a double long splint with transverse bars; but a Bradford frame is better, and Whitford's modification of the Bradford frame probably best of all. The quality of the nursing is, however, more important than the form of the frame. This treatment is generally very successful, but it must be persevered in for a year at least; and often eighteen months

or two years may pass before the child can be safely allowed to get up and go about in a stiff jacket. During the treatment the patient should be kept in the fresh air and sunshine as much as possible, and should sleep in an open-air shelter. It is well to ascertain the progress of the healing process in the vertebræ by an X-ray examination before he is allowed any increase of movement.

Spinal Muscular Atrophy of Infants (*Infantile Progressive Muscular Atrophy* (*Werdnig-Hoffmann Paralysis*),¹ *Amyotonia Congenita* (*Oppenheim's Disease*)).—This is a rare form of progressive paralysis present at birth or commencing soon afterwards and due to degeneration of the spinal motor cells. It affects the sexes equally and may show a familial tendency.

Attempts have been made to classify the cases into two separate groups according to whether the disease is familial or not, is congenital or only develops later, and whether or not there is a tendency to improvement. The customary differential diagnosis is as shown in the accompanying Table.

<i>Werdnig-Hoffmann Paralysis.</i>	<i>Oppenheim's Disease.</i>
Acquired.	Congenital.
Familial.	Not familial.
Paralysis progressive.	Paralysis diminishing.
Atrophy of muscles apparent.	Atrophy of muscles masked.
Localised atrophy commencing in pelvic region.	General hypotonia.
Reflexes proportionate to atrophy.	Reflexes absent.
Reaction of degeneration present.	Reaction of degeneration absent.
Atrophy of spinal cells.	No spinal lesion.

It has been shown by several writers that such a classification is untenable. Greenfield and Stern² have pointed out that paralysis of the Werdnig-Hoffmann type is not always progressive but that it may come to a standstill or improve, and as a further similarity between the two groups is the fact that there is never any implication of the diaphragm or involvement of the cranial nerves. Paterson³ has published a series of six cases (in all of which there was evidence post-mortem

¹ G. Werdnig, *Arch. f. Psychiatrie*, xxii., 437; J. Hoffmann, *Arch. f. Psychiatrie*, xx., Heft 3, and *Deutsche Zeitschr. f. Nervenheilk*, iii., 427; J. Thomson and A. Bruce, *Edin. Hosp. Rep.*, 1893, i., 361.

² J. G. Greenfield and R. O. Stern, *Brain*, 1927, l., 652.

³ D. Paterson, *Westminster Hosp. Rep.*, 1929, xx., 43.

of degeneration of the spinal motor cells) which reveals very clearly the variability in time of onset (four of the six were congenital) and in the question of familial tendency (two of the six cases were clearly familial), two points supposed to differentiate the two groups. As a search of the literature reveals a dearth of histological evidence of the primary muscular dystrophy, and as several cases¹ with definite spinal lesions have been classified as Oppenheim's Disease, there would seem no justification for considering Werdnig - Hoffmann Paralysis and Oppenheim's Disease as distinct entities.

The disease then is one which may or may not present a familial tendency. The muscular weakness is usually present at birth, but it may develop later. Sometimes the child has begun to walk a little before the symptoms are noticed.

The paralysis and muscular wasting are symmetrical in distribution and, though this commences in the region of the pelvic girdle, all muscles of the limbs and trunk are ultimately involved. The muscles of the forearm and hand, in contrast to the pure myopathies, do not escape (Fig. 228). There is, however, no paralysis of the cranial nerves, of the diaphragm, or of the sphincters, and the mental condition is unaffected. Sensation is not impaired but cramp-like pains sometimes occur, and passive movements of the limbs may be painful. The deep reflexes are weak or absent. The affected muscles do not as a rule react to the faradic current, and the reaction of degeneration is often present in the later stages.

At first the child is well nourished, but towards the end there is great emaciation, and contractures of the limbs. The condition progresses steadily in spite of treatment, and death occurs in a few years (one to five), usually as the result of a pulmonary complication.

The little girl shown in Figs. 227 to 229 was normal at birth and only began to lose strength after the end of the first year. The paralysis and muscular wasting advanced steadily but very slowly. When four years and eight months old, she had an attack of bronchitis, in the course of which almost complete collapse of the left lung occurred. After a few days of severe dyspnoea the respiration improved; and she lived for eight months longer in a state of increasing emaciation and debility.

¹ J. B. Holmes, *Amer. Journ. Dis. Child.*, 1920, xx., 405.



FIG. 227.—Child of 4 years and 4 months. Shows state of nutrition, characteristic attitude, and hyper-extension of fingers on the attempt to spread them out.



FIG. 228.—Same child at 4½ years. Shows emaciation, deformity of chest from collapse of lung, presence of lower part of pectoralis, and equable distribution of wasting in arms and hands.



FIG. 229.—Child of 5 years and 10 months. Shows extreme wasting, and distortion of lower limbs from contracture of muscles.

Peroneal Muscular Atrophy (*Charcot-Marie-Tooth Type of Muscular Atrophy*).

This is a condition analogous to the above (*Werdnig-Hoffmann Paralysis*) in that it also is familial in incidence and due to degeneration of the spinal motor cells. The age of onset, however, is different, being the fifth to the tenth year, or even later. The mischief is limited to that part of the spinal cord governing the limbs, and especially the distal portions of the limbs, and the course is much longer, extending over as long a period as twenty years. Boys and girls are equally susceptible.

The disease makes its appearance in the feet, the intrinsic muscles of which become atrophied, with retraction of the toes and the development of *pes cavus*. Slowly the atrophy extends to the anterior tibial and peroneal groups, in which fibrillary twitching is often observed. One characteristic feature is that the muscles are seldom attacked in their whole length; thus the distal half may be severely wasted while the proximal half appears well developed. When the disease extends above the knee, owing to this partial atrophy of the muscles, the thigh has the appearance of an inverted champagne bottle. In some cases the hands and forearms become affected, but the muscles of the trunk and the sphincters remain intact. The atrophied muscles become fibrosed and the consequent contractures are often sufficient to hold the joints fixed as if by splints.

The plantar reflexes and the ankle-jerks are absent, but the knee-jerks are present and brisk. The electrical excitability of the muscles is diminished, and polar changes (*Reaction of Degeneration*) may be present. Sometimes sensation is involved.

The only *treatment* consists in sustaining the general health and the application of splints to make walking possible, but, as previously mentioned, this may occur naturally in consequence of the muscular contractures. Thyroid gland extract has been recommended.

Cerebral Paralysis.

Paralysis from a brain lesion may arise under many conditions. It is met with as one of the symptoms of tuberculous meningitis, of tumour, of abscess, and of syphilitic and other diseases of the brain. There are also a number of cases of cerebral hemiplegia

which, although they may be due to different lesions, resemble each other closely in their symptoms.

Infantile Cerebral Paralysis.—As their cause cannot usually be determined during life, these cases are best referred to as infantile cerebral paralysis or infantile hemiplegia—the paralysis being nearly always unilateral.

According to Osler,¹ “Infantile hemiplegia is commonly the result of a variety of different processes, of which the most important are—

“1. Hæmorrhage occurring during violent convulsions or during a paroxysm of whooping-cough.

“2. Post-febrile processes: (*a*) embolic; (*b*) endo- and peri-arterial changes; and (*c*) encephalitis.

“3. Thrombosis of the cerebral veins.”

It seems probable, as Strümpell long ago pointed out, that many of the acute cases of infantile hemiplegia are the result of *polioencephalitis* due to the same infection as ordinary poliomyelitis. A number of them, however, especially those due to vascular lesions, occur in the course of infectious illnesses such as measles, scarlet fever, or diphtheria, or follow toxic diarrhœa.

The onset of the *symptoms* in these cases is generally sudden, and usually, although not always, accompanied by convulsions or coma. Often there is considerable rise of temperature, and occasionally vomiting. Rarely, the paralysis is slight at first, and after repeated convulsive attacks becomes more complete; generally, however, it is complete from the beginning. The face is often, but not always, affected. After the acute symptoms have lasted a varying number of days, they gradually subside and the extent of the paralysis becomes evident. At first the paralysed limbs are flaccid, but in a few days rigidity sets in. In some cases the paralysis gradually passes off, but, in most of them, a more or less severe degree of rigidity and powerlessness is left.

The arm is generally more completely paralysed than the leg, and the forearm becomes fixed in the prone position (Fig. 230). The muscles are not usually much atrophied, but they are in a state of more or less rigid contracture. The leg recovers to a greater extent than the arm, and occasionally, as already mentioned, no trace of the paralysis remains.

¹ W. Osler, *The Cerebral Palsies of Children*, 1889, 96.



FIG. 230. — Infantile Cerebral Paralysis. (Boy of $2\frac{1}{2}$ years.)

The deep reflexes are almost invariably increased. Sensation is usually quite unaffected. In a considerable proportion of cases, choreic or athetotic movements develop later. Aphasia is quite often found in cases of left as well as in those of right hemiplegia, but it is never permanent. Mental defects and eccentricities are common. Epileptiform seizures often occur; they may be of the nature of *petit mal*, or general convulsions with unconsciousness, but sometimes they consist in unilateral spasms without loss of consciousness.

The *diagnosis* of the lesion is usually exceedingly difficult at first. It is generally, however, tolerably easy to distinguish between them and cases of poliomyelitis. The main points of difference are as given below by Sachs.¹

ACUTE SPINAL PARALYSIS.

Onset sudden with fever, coma, and convulsions. Convulsions rarely repeated after first few days.

Paralysis flaccid, associated with atrophy.

Paralysis widely distributed, possibly involving all extremities, or narrowly limited to one member, or even a single group of muscles.

Electrical reactions altered (R. D.).

Deep reflexes diminished or lost.

Intellect never permanently involved; no epilepsy.

ACUTE CEREBRAL PARALYSIS.

Onset sudden with fever, coma, and convulsions. Convulsions apt to be repeated.

Paralysis spastic; no atrophy; associated with rigidity and contractures.

Paralysis generally hemiplegic, sometimes diplegic or paraplegic; monoplegia rare.

Electrical reactions normal.

Deep reflexes exaggerated.

Intellect often involved; epilepsy frequent.

The *prognosis* is almost always unfavourable. In rare cases, especially those due to hæmorrhage (such as occasionally occurs during an attack of purpura), there may be a complete recovery of power. In most cases, however, only slight improvement, if any, is observed. In some, even when convulsions have not been present at the onset, the damage to the brain predisposes to

¹ B. Sachs, *The Nervous Diseases of Children*, 1895, 305.

their later occurrence; and in cases where the intellect is not seriously affected from the first, there is apt to be a tendency to mental defect or peculiarity in later life.

This form of paralysis, after the first few weeks, should be *treated* mainly by active and passive exercises. If energetically persisted in, these always lead to some improvement. Massage is of no use; it is with stiffness and lack of control of the muscles and not with weakness that we have to do; passive movement to overcome the contractures may, however, be practised.

In this class of diseases, as well as in infantile spinal paralysis, great improvement sometimes results from suitable surgical measures.

Intracranial Tumours.

More than half of the intracranial tumours in childhood which produce symptoms are of the nature of caseating tuberculous masses. Of 25 children, varying in age between fifteen months and ten years, admitted to the R.H.S.C., Glasgow, with characteristic symptoms, and in whom post-mortem examination permitted of a diagnosis of the nature of the tumour, 15 were tuberculomata, 7 gliosarcomata, and 3 gliomata. This even does not give a true idea of the frequency of tuberculomata, as the majority of tuberculous nodules in the brain in early life are quite latent—they produce no symptoms and are often only discovered after death, which in many instances is the result of tuberculous meningitis. Of 71 intracranial tumours discovered post-mortem, in the majority of instances accidentally, 58 were tuberculous in nature. Other types of cerebral tumour found in childhood are sarcomas and endotheliomas (suprapituitary): gummata are extremely rare.

There is a marked difference between the usual position of intracranial tumours in children and in adults (James Taylor).¹ In early life the great majority are situated below the tentorium; while after sixteen the conditions are reversed, and supratentorial are much commoner than infratentorial tumours. Of the above-mentioned 25 examples which came to post-mortem examination, 15 were situated in the cerebellum, pons, or medulla, and hence below the tentorium. This does

¹ J. Taylor, *Nervous Diseases in Childhood and Early Life*, London, 1905, 165.

not apply to the latent tuberculous nodules, which are often found in the cerebral cortex in infants.

Boys and girls seem equally subject to the condition. Of 160 cases, 86 were boys and 74 girls.

Though cerebral tumour may be met with at all ages, it is uncommon during the first two years. The age distribution in 160 examples is given in Fig. 231.

The **causation** of intracranial growths is quite obscure, but they seem in some cases to begin after an injury.

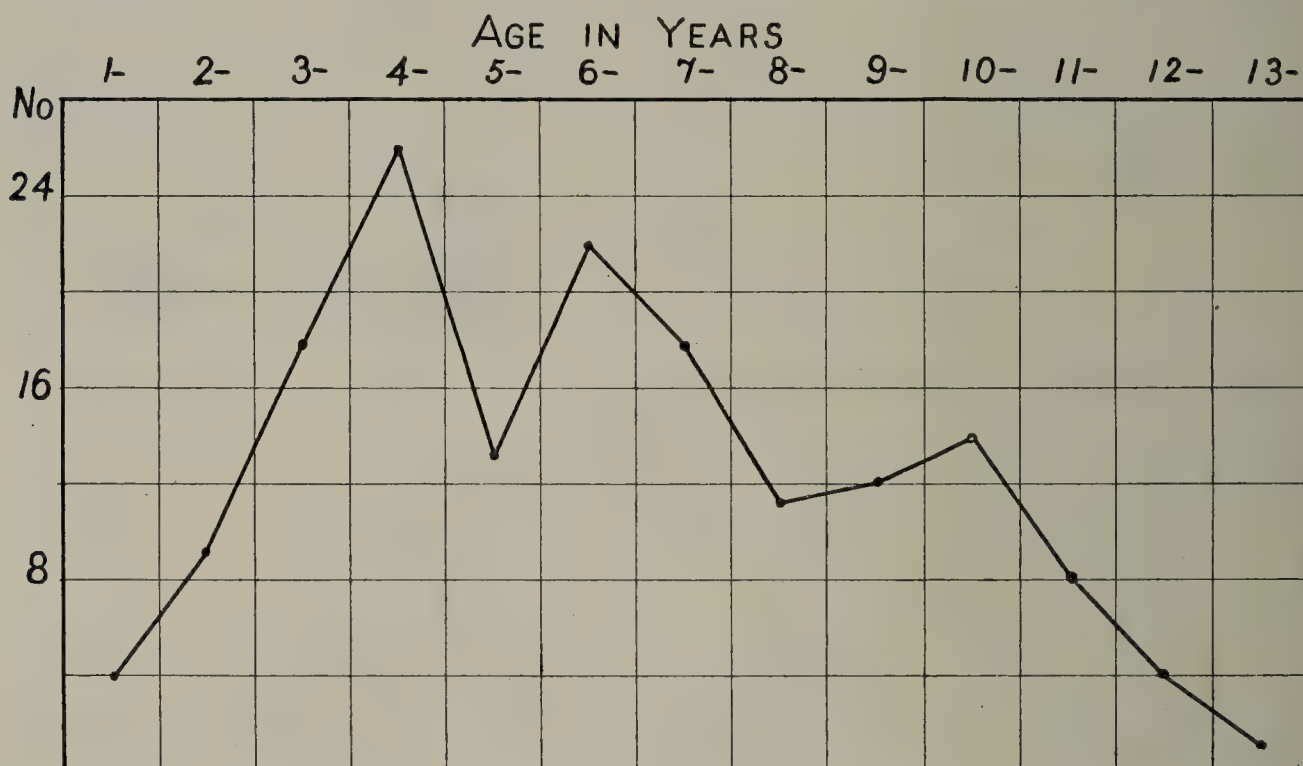


FIG. 231.—Age Incidence of Cerebral Tumour in 160 cases (R.H.S.C., Glasgow).

Symptoms.—The three cardinal symptoms of intracranial tumours—*headache*, *vomiting*, and *optic neuritis*—are generally present. Headache is a fairly constant feature, though it is relatively less severe in children than in adults. Vomiting and optic neuritis are sometimes both absent. The occurrence of general *convulsions* may be the first indication of the presence of a tumour, and the attacks may go on recurring for a long time without any localising symptom appearing. They may, however, occur at various stages of the disease. *Nystagmus*, *giddiness*, and *mental dullness* are also common; and secondary *hydrocephalus* often sets in, especially if the tumour is so situated as to obstruct the aqueduct of Sylvius or the fourth ventricle. When the hydrocephalus is severe the cranial sutures become separated (Fig. 232) and tapping of the skull elicits the cracked-pot sound. This is best obtained by giving

the skull a sudden tap with the end of the middle finger in the region of the lambdoidal suture. This starting of the sutures with the cracked-pot sound is, in our experience, only present when the tumour is subtentorial and hence has a definite localising value. It would seem highly probable that this splaying of the sutures by relieving the pressure is responsible for the mildness of the pressure symptoms, *e.g.*, headache and optic neuritis, so characteristic of the condition during childhood.

Although in most cases of intracranial tumour in children



FIG. 232.—Skiagram of skull in child suffering from Subtentorial Cerebral Tumour. Note widening of the sagittal and lambdoidal sutures and "paw-feet" markings in frontal and occipital regions.

only general symptoms are present, in others localising symptoms also occur; but in young children these are often very difficult to make sure of.

Lesions of the hemispheres are less likely to produce localising symptoms in children than in adults. This is because the functions of the various areas of the cortex are as yet incompletely differentiated, and are therefore more ready to take up the work of neighbouring parts when these are damaged by disease. One result of this compensatory action of the other cortical areas is that aphasia is almost unknown in children as a symptom of cerebral tumour.

Cerebellar tumours are specially important as they are so common in childhood. They give rise to the same symptoms as in later life, such as vomiting, headache, optic neuritis, and ataxia of the side next the lesion, with normal deep and superficial reflexes. The attitude of the head which is found in these cases is often peculiar and characteristic (Fig. 233).

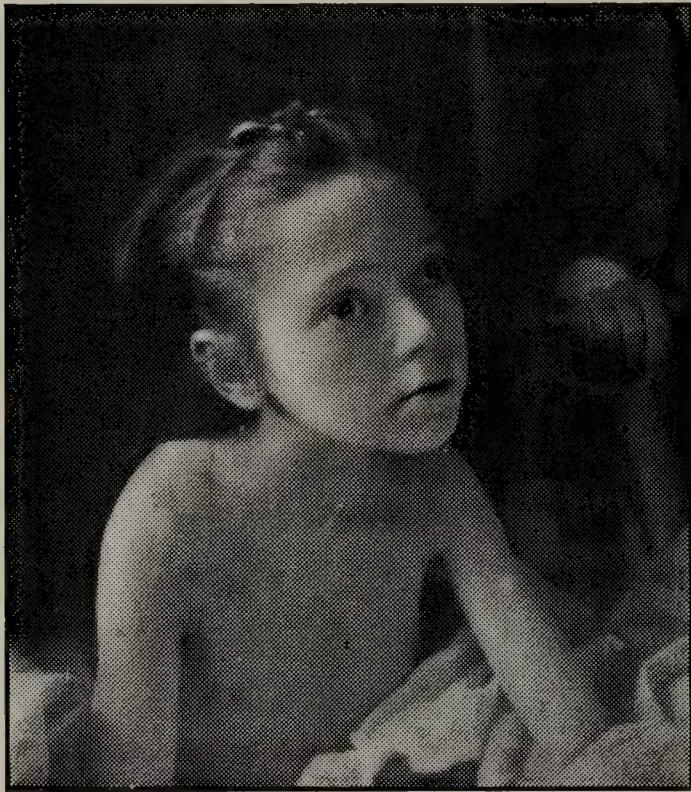


FIG. 233.—Attitude of head in Cerebellar Tumour.

Batten describes it as follows: "The ear on the side of the lesion is approximated to the shoulder of the same side, and the side of the face corresponding to the cerebellar lesion is turned downwards, and the chin is diverted to the opposite side." It is characteristic of the symptoms of cerebellar tumours that they vary considerably in severity from time to time. Of all the tumours during childhood that situated in the crus, causing crossed paralysis, is the most characteristic.

The **diagnosis** of cerebral tumour may be one of the easiest of tasks but, on the other hand, it may be one of the most difficult. Cerebral tumour should always be considered in any anomalous mental condition. *Headache, vomiting, and giddiness*, with *optic neuritis*, may all be present together or they may be present in various combinations. It has already been stated that when the increased intracranial pressure is great, as in cerebellar tumour, splaying of the cranial sutures occurs, and this is a most valuable sign, not only in deciding that a tumour is present, but in pointing to its localisation below the tentorium. The mistake most often made is to regard the facial or other paralyse of commencing tuberculous meningitis as localising signs of cerebral tumour. But even in the presence of meningitis a severe degree of optic neuritis, or a neuritis apparently passing off, with a marked cracked-pot sound, would suggest a pre-existing tumour as the origin of the meningeal mischief.

It is of some importance to form an opinion regarding the nature of the growth because of its effect on our line of treatment. A positive Wassermann reaction would be in favour, but not absolutely diagnostic of a gumma. Although a positive tuberculin reaction (Mantoux) is little indication that the tumour is of the nature of a tuberculoma, a negative tuberculin reaction, on the other hand, is almost conclusive proof that it is not tuberculous.

The *localisation* of cerebral tumours in childhood is a matter of the greatest difficulty. In the first place, children are not able to describe their symptoms with any degree of precision, or to co-operate in the various manœuvres and tests useful in localisation. When we appreciate, too, that the majority are tuberculomata and consequently multiple, the difficulty in localisation is easily understood. Nevertheless, those situated in the cerebellum, in the cerebello-pontine angle, and in the crus and upper part of the pons, because of the characteristic signs, can often be recognised. Tumours of the suprapituitary fossa, because of the tendency to show calcification, the accompanying hemianopia, and the X-ray appearances of the sella turcica, may be readily recognised: this type of tumour, however, is not common during childhood. X-ray examination, unless after the introduction of air into the ventricles, as first performed by Dandy, is of little value in revealing the situation of the tumour, though from the splaying of the sutures and the "paw-feet" appearance of the inner surface of the calvarium the presence of tumour may be surmised (Fig. 232). With the introduction of air into the ventricles, however, it is held that much information in this direction may be obtained.

The **prognosis** of intracranial tumours is almost always extremely bad; but the course of the disease varies in different cases. It, of course, greatly depends on the nature of the growth. Sometimes in tuberculous and gliomatous tumours it may be very chronic, and—with or without treatment—temporary arrest of the symptoms is not uncommon. Occasionally, cases—especially cerebellar cases—about the diagnosis of which there seems no doubt, entirely recover. In gliomata the symptoms are usually more rapidly progressive and more severe, and hæmorrhages into the substance of the growth are apt to occur. Cases of tuberculous tumour often end in tuberculous meningitis.

Treatment.—Unless the tumour is of a gummatous nature the administration of drugs cannot be expected to have any effect.

In the case of tuberculomata their tendency to be multiple makes them little suitable for attempted removal. Any interference, too, is apt to precipitate a meningitis. They may, however, like other tuberculous lesions, undergo healing, and this may be rendered more probable by general open-air treatment as employed in other forms of the disease. Tumours of the suprapituitary fossa (adenoma) have within recent years shown themselves specially suitable for removal, as also those (neuromata) of the cerebello-pontine angle. Cerebellar tumours are often difficult of removal, and of course those situated at the base (in the basal ganglia, the crus, the pons, and medulla) are beyond the help of surgery.

Although removal of the tumour itself is so often outwith the range of the possible, relief may be given to the pressure symptoms (headache, vomiting and papilloedema) by decompression. This operation is best performed in the temporal fossa without opening the dura, which, with the strong temporal muscle, sufficiently supports the brain and hinders the development of a cerebral hernia. It is generally held that whenever any degree of papilloedema appears decompression should be carried out in order to save the eyesight from the consequent post-papillitic optic nerve atrophy. During the height of the papilloedema the eyesight may be unimpaired and it is only when atrophy sets in that this fails. Whether the operation of decompression saves the eyesight or not, it usually gives considerable relief to the symptoms, and in some cases would appear to bring about an arrest of the growth.

CHAPTER XXX

ACUTE POLIOMYELITIS AND ENCEPHALITIS LETHARGICA

Acute Poliomyelitis (*Infantile Spinal Paralysis*).

THE first thorough descriptions of acute poliomyelitis were published in 1840 by v. Heine¹ and in 1887 by Medin,² and on the Continent the disease is often called after these observers (Heine-Medinische Krankheit). Much literature has since appeared on the subject, an excellent summary of which will be found in F. E. Batten's "Lumleian Lectures."³ During the last few years the extensive epidemics, in America and elsewhere, have produced a large amount of valuable literature.⁴

Epidemiology.—Until towards the end of last century the disease was regarded mainly as one which occurred sporadically; but, even in 1840, Heine noted its occasional occurrence as an epidemic and suspected its possible spread by infection. In 1868 there was an epidemic in Norway.

The first epidemic, however, to be fully recorded was one in Sweden in 1881. In 1883 small groups of cases were observed in Norway, Germany, and France, and in 1887 Medin gave a full account of a small outbreak in Stockholm. During the "nineties," small epidemics were reported in Italy, France, Australia, England, America, Austria, and Sweden (Wickman⁵). Between 1900 and 1910 the disease occurred in large epidemics in various parts of Europe, in North and South America, in Australia, and in the South Sea Islands. In these, the numbers affected ran into four figures; while, in

¹ Jac. v. Heine, *Spinale Kinderlähmung*, Stuttgart, 1840.

² O. Medin, *Proc. 10th Internat. Med. Congr.*, 1891, ii., div. vi., 37.

³ *Brain*, 1916, xxxix., 115.

⁴ G. Draper, *Acute Poliomyelitis*, London, 1917; and J. Ruhräh and Erwin E. Mayer, *Poliomyelitis in all its Aspects*, Philadelphia and New York, 1917.

⁵ Wickman, *Beiträge zur Kenntniss der Heine-Medinische Krankheit*, Berlin, 1907.

the former outbreaks, a few dozen cases at most had been reported.

In 1904 Wickman made the very important observation that the disease often occurred in an abortive, non-paralytic form, and that these cases, being usually unrecognised, were possibly a common cause of the spread of the disease.

Between 1903 and 1907 poliomyelitis seems to have been pandemic in Scandinavia; and from 1907 to 1910 large epidemics occurred in New York State and in Massachusetts.

In this country the prevalence of the disease has hitherto been mainly sporadic, but it has not been very rare to meet with two or even more cases in one family. Occasionally small epidemics have been reported. One of the more severe of these, in which sixty-two cases were collected, occurred in Edinburgh and the surrounding counties in 1910.¹

As further and larger outbreaks will probably occur in this country, it is important to study the spread of the disease and such factors as have been common to previous epidemics.

Seasonal Incidence.—In the Northern Hemisphere the disease appears mainly between July and October; in the Southern it is generally met with in March and April. In the tropics its time of incidence has varied; it occurred in Cuba in 1909 in July and August, and also, very severely, in an island in the South Pacific in January of the same year.

Age Incidence.—Poliomyelitis is rare in early infancy. It mainly affects children during the second and third years; the numbers diminish steadily in later childhood, and cases in adults are usually comparatively rare, although in some epidemics in rural areas a large proportion of the patients have been adults.²

Mortality.—This varies in different epidemics, and also according to the extent to which abortive cases are included in the statistics. It is said to be, on an average, about 11 to 12 per cent.; but in a recent New York epidemic it was 27 per cent., even although the statistics included more non-paralytic cases than those of any former epidemic (Draper). The mortality is higher in adults than in children.

The duration of the *incubation* shows considerable variations. In the human being it generally lasts less than four days, and

¹ H. Bruce Low, *Proc. Roy. Soc. Med.* (Epidemiol. Sect.), 1912, vol. v., 76.

² W. Wernstedt, *Ergeb. d. inn. Med. und Kinderh.*, 1924, xxvi., 306.

sometimes as much as a week or even more. A fortnight should be allowed for quarantine. In experimental animals it is usually from eight to ten days, but it may be much longer.

Spread of Infection.—Wickman showed that the spread of the disease by carriers usually follows the lines of communication (roads and railways), and that it is favoured by school attendance. The carriers may be entirely free from disease of any kind.

Experimental Work.—Great advances in our knowledge of the subject have resulted from experimental investigation.¹ In 1908 Landsteiner and Popper succeeded in infecting a monkey by the injection of substance from a human spinal cord. In the following year Flexner, Lewis, and various continental observers showed that the disease could be transmitted from one monkey to another by injection of an emulsion of a diseased spinal cord. In 1913 Flexner and Noguchi were able to cultivate its apparent cause²—an ultra-microscopical organism, which retained some of its virulence even after it had been passed through the finest known filters. They found that it could be preserved in glycerine in a state of activity for many months, that the infection was able to penetrate the uninjured mucous membrane of the nose and bowel, and that flies could transmit it. The finality of Flexner's conclusions has since been questioned, and the matter is still *sub judice*.

Pathological Anatomy.—The lesion of poliomyelitis is present for the most part in the anterior horns of the spinal cord where there is the characteristic accumulation of mononuclears in the perivascular lymphatic spaces—the so-called cuffing of the vessels. There are often present, in addition, hæmorrhages and the motor cells are degenerate. Congestion of the meninges is also present, and it is this which accounts for the changes in the cerebro-spinal fluid.

Although the principal lesions are found in the nervous structures, less severe changes have been described in other viscera. These include hyperplasia of the lymphatic glands and spleen, changes in the bone-marrow, interstitial small-cell infiltration in the portal spaces in the liver, and general cloudy swelling of the organs.

¹ F. E. Batten, "Polioencephalitis and Poliomyelitis," *Trans. 17th Internat. Congr. Med., London, 1913*, sect. x. (Diseases of Children), 145.

² *Journ. Exper. Med.*, Oct. 1913, xviii., 461.

Symptoms.—Although the first indication of the disease may be paralysis of one or more limbs, in many instances the onset of the paralysis is preceded by a period of fever and indefinite malaise. Hence it is customary to speak of a *pre-paralytic* and a *paralytic stage* of the disease.

In the *pre-paralytic stage* there may be fever, vomiting, and headache, with generalised pains and some nuchal rigidity. The symptoms at this stage, unless during an epidemic, are usually looked upon as influenzal in nature or due to a chill. An examination of the blood will, however, disclose a leucocytosis, thus eliminating influenza, and if lumbar puncture is performed the cerebro-spinal fluid will be found under increased pressure, not absolutely clear, with an increase in the number of cells (30 to 2000 per cubic millimetre) and in the amount of globulin. At first the cells may be chiefly polymorphonuclear, but soon they are lymphocytic in nature. The globulin content is most marked later in the disease when the cells have greatly diminished or disappeared. The glucose and chloride contents of the fluid are normal. It is important to recognise the disease in the pre-paralytic stage, as it is only at this time that serum treatment (p. 764) is of any avail.

The duration of this period varies much in different cases. In the following Table (after Aycock and Luther) is shown the time of onset of the paralysis after the commencement of the illness, as occurred during an epidemic.

Time of Appearance of Paralysis (Aycock and Luther).¹

		Day of disease on which paralysis appeared.									
		1	2	3	4	5	6	7	8	9	10
No. of Cases	.	25	78	148	182	72	40	16	13	7	6
Per cent.	.	4	13	25	30	12	7	3	2	1	1

The *paralytic stage*, as already mentioned, may appear apparently unheralded by any fever or malaise. The child goes to bed at night in perfect health, and in the morning is found to be paralysed in one or more limbs. Occasionally the mother reports that the child had been somewhat restless in his sleep, but at other times absolutely nothing abnormal had been noted.

¹ W. L. Aycock and E. H. Luther, *Journ. Amer. Med. Assoc.*, 1928, xci., 387.

A *clinical* classification has been proposed by Draper, in which the cases are divided into three groups, according to the nature of their onset and their clinical course. In one of these—the *sudden onset group*—the meninges and nervous tissues are evidently affected from the first. Another he calls the *dromedary group*, because in them an initial rise in temperature with general symptoms is followed, after an intervening apyrexial interval, by a second rise or “hump” with nervous manifestations. These cases were formerly described by Sir Thomas Barlow as “jump cases.” In the third and last type—the *straggling group*—the symptoms are like those of the dromedary cases, except that in them there is no intervening interval of well-being between the first and second acute periods.

A useful pathological classification is that suggested by Wickman,¹ according to whether the symptoms indicate chiefly involvement of (1) the *spine*; (2) the *medulla, pons, and mid-brain*; (3) the *cerebrum*; (4) the *cerebellum*; (5) the *meninges*; (6) the *nerves*; and, lastly, there is a large group of *abortive cases*, in which there is no paralysis at all. Such a classification is certainly useful, although it may on some grounds be open to criticism.

(1) *The Spinal Form*.—As the spinal cases are much commoner than those of all the other paralytic groups put together, we may begin by describing the symptoms of an ordinary spinal case of the *sudden onset type*.

In many of the cases we are told that the child has gone to bed quite well and has wakened in the morning unable to move a leg or an arm. Often, however, we find, on close inquiry, that for a day or two before this occurred there have been slight indications that the child was not quite as well as usual. In the commonest and most typical of these cases, the onset differs little, at first sight, from that of most other acute infectious diseases. In the midst of perfect health, the child becomes feverish, drowsy, and restless, and complains of headache; the temperature goes up to 101° to 103° F., and there may be vomiting or diarrhoea, a sore throat, or a convulsion. Although the general symptoms are so like those of other feverish ailments of childhood, there is often, as Draper points out, a peculiar character in the symptoms which

¹ F. E. Batten, *Brain*, 1916, xxxix., 115.

may suggest the real nature of the attack to those who have seen many cases. The eyes look somewhat puffy and have a glazed appearance, and the patient is nervous and very irritable; he is extremely anxious to be left alone and resents being touched or even spoken to. A very characteristic



FIG. 234.—Poliomyelitis of Right Arm.

symptom consists in tenderness on any forward movement of the spine. This leads the child to keep his back stiff, and to resent strongly any attempt to bend the neck or body. Along with this there is often a well-marked Kernig's sign with resistance and tenderness on movement of the limbs, and sometimes a definite degree of opisthotonos develops. This stiffness occurs before there is any implication of the meninges. Draper suggests that the pain on bending the spine may be due to the intervertebral spinal ganglia being pulled on by this movement. On physical examination, nothing definite is found except a furred tongue and some redness of the fauces. Apart from the general irritability and stiffness, there is usually no sign of the nervous system being affected at this stage.

The spread of the disease to the meninges is indicated by severe headache, or by disturbance of the tendon reflexes. Occasionally there is swelling of the joints of the affected

limbs, and transitory erythematous or other rashes may be present.

The paralysis, in the great majority of cases, is complete at an early stage of the illness in this type—within the first twenty-four or forty-eight hours, or at latest by the third or fourth day. After a few days or weeks, there is generally more or less noticeable recovery of power in some of the muscle groups affected. Within about two months of the beginning of

the attack, marked wasting of the affected limb is evident, with lowering of its temperature; and, within a week, the reaction of degeneration may be demonstrated. When the paralysis is severe and extensive, the whole of the affected limb, including the bones, often ceases to grow at the same rate as the others.

The *distribution* of the paralysis varies in different cases. Most frequently the disease affects one lower limb only, but often both are paralysed. Sometimes all the limbs are affected, sometimes one arm and one leg, and sometimes one arm only (Fig. 234). It is rare for the arm and leg of the same side to be affected. In some cases there is marked implication of the muscles of the neck and back, and occasionally those of the abdomen are severely paralysed. When this occurs and the weakness persists, a peculiar bulging of the abdominal wall occurs when the child cries (Fig. 235). In a few cases the facial muscles on one side are paralysed along with one or



FIG. 235.—Poliomyelitis of Abdominal Muscles. Showing bulging during crying.

more limbs, and occasionally the face is the only part affected. This form of facial paralysis practically always recovers rapidly.

The other types are all much rarer than the sudden onset type.

In the "*dromedary type*" of case, as already mentioned, the

first period of fever is followed by an apyrexial interval, which is succeeded by a second rise of temperature. The name is now also applied to cases in which the two periods of acute symptoms are unaccompanied by corresponding "humps" on the temperature chart. The duration of the "humps" varies from a few hours to several days, and their severity also varies greatly—being extremely slight in some instances and in others severe. The symptoms during the second "hump" in this type are due to the involvement of the nervous system, and correspond exactly to those during the onset in the sudden onset cases in which the virus has found its way rapidly into the nervous tissues. Those during the first "hump" are caused by the general systemic infection, including probably infection of the spleen and bone-marrow. When they are mild in character they are often overlooked or forgotten by the parents.

Following the complete remission of the symptoms in the first group or their varying duration in the second, the patient may go on to recovery, or he may suddenly develop signs of involvement of the nervous system; and, when this occurs, the case may end either in recovery or in continued paralysis.

In the "*straggling type*" of cases the onset and course of the symptoms are the same as in the group just described, the only difference being that in them there is no apyrexial interval between the "humps." The original dose of the virus has either been larger or more intense, or else the child's resistance has been lower, so that the indications of a general infection continue until the nervous system has become affected.

The other five paralytic varieties of acute poliomyelitis are all more or less rare. They may occur in an uncomplicated form, but are often accompanied by spinal symptoms.

(2) *The Bulbar and Pontine Form.*—In this, any of the cranial nerves may be involved, especially the facial.

(3) *The Cerebral Form.*—When the brain is affected the symptoms vary according to the part of the hemisphere implicated. If the frontal region has been attacked, the child shows symptoms of severe mental deterioration such as senseless screaming, failure to recognise his parents, dirty habits, and unintelligible speech. If the disease is in the motor areas, hemiplegia or diplegia may be produced; and if the occipital cortex is the part involved, cerebral blindness follows, which may or may not be recovered from.

(4) *The Cerebellar or Ataxic Form.*—This leads to ataxia of the cerebellar type which sets in with cerebral symptoms, and may either be unilateral or involve both sides. The intellect is not affected, and there is no loss of muscular power. Nystagmus may be present. These symptoms, like those of the other forms, may be permanent or may recover entirely.

(5) *The Meningitic Form.*—In some cases of poliomyelitis there are symptoms which are indistinguishable from those of meningitis—such as headache, coma, head retraction, Kernig's sign, and convulsions—and the characters of the cerebro-spinal fluid also suggest involvement of the meninges. Complete recovery occurs sometimes after a short illness.

(6) *The Neuritic Form.*—Some writers on infantile paralysis have been of opinion that a few of the cases that looked like poliomyelitis may have really been caused by affection of the spinal nerves. This seems quite possible, but it has not yet been proved. Occasionally instances of evident neuritis have occurred, along with cases of poliomyelitis in other members of the same family.

(7) *The Abortive or Non-Paralytic Form.*—As has been already mentioned, Wickman drew attention in 1904 to this very important type of case; and the experience of recent epidemics has shown that it is quite common. Draper estimates the proportion of such cases as 50 per cent. of the whole number, and others have placed it even higher. The children affected present exactly the same general symptoms as occur during the early stages of the ordinary paralytic cases, but without any paralysis following. The character of these general symptoms varies in the non-paralytic, as in the paralytic, cases. In one group they are mainly gastro-intestinal, in another connected with the throat, while in a third the patient may only show general malaise with no local manifestations. When such cases occur sporadically, they are usually mistaken for influenza. It is, of course, obvious that they may possibly constitute a serious cause of the spread of the disease.

Seeing that the central lesion in *herpes zoster* is so similar to that in acute poliomyelitis, it might be expected that the two diseases would often occur together; but this has only rarely been described. During the Edinburgh epidemic, already referred to, there were no more than the usual number of cases of herpes zoster brought to the Children's Hospital.

Diagnosis.—The presence of poliomyelitis can only be recognised before the paralysis develops from an examination of the spinal fluid. The disproportion between cell increase and that of the globulin is characteristic. The increase in globulin appears after that of the cells and there is a normal chloride content, thus differentiating it from tuberculous meningitis (p. 777).

When a local loss of power sets in acutely, however, with flaccidity of the muscles and the other clinical characters already described, the diagnosis is easy, and the early onset of muscular wasting soon supplies its confirmation.

There may be some difficulty in the early stages in distinguishing the meningitic and cerebral types of the disease from cerebro-spinal meningitis. This difficulty can usually be removed by an early examination of the cerebro-spinal fluid; and the fact that cerebro-spinal meningitis prevails mostly in the spring, and poliomyelitis in the late summer and autumn months, is also helpful. After a few days, if any local paralysis has appeared and is at all extensive, the diagnosis becomes simple. When the muscular involvement is very limited in extent, however, it may be difficult, or impossible, to make sure of it in the early stages of the case.

Occasionally, cases of rachitic, syphilitic, and scorbutic pseudo-paralysis, and of acute infective epiphysitis and rheumatism, have been mistaken for the results of poliomyelitis. We have also known both diphtheritic paralysis of the lower limbs and paresis of an arm occurring during the onset of chorea to be mistaken for this disease. In these cases the paralysis always begins gradually.

Old-standing cases of poliomyelitic paralysis affecting small muscular areas in the lower limbs sometimes show defective movements which are apt to be attributed to congenital malformations or to hip-joint disease. The diagnosis between spinal and cerebral paralysis in childhood has been dealt with in the last chapter (p. 746).

Prognosis.—In the acute stages of the disease the prognosis as to life is good, except in those cases in which the respiratory muscles are involved. Death never occurs from toxæmia. When a child recovers from an acute attack with his muscles of respiration much affected, the risk of death from any chance catarrh spreading to the bronchi and lungs, at a later period, is very serious.

During the attack the prognosis as to its probable effect on the child's future cannot be given with any certainty, until sufficient time has elapsed to make sure that there is not going to be a further extension of the lesion. In nearly all the cases, if no additional weakness occurs within six to twelve hours of the first paralysis, there is little chance of any further involvement; but a later spread is just possible, up to the seventh or eighth day.

According to Draper's experience, profoundly stuporous cases generally recover; and he believes that "increased alertness and apprehension on the part of the child with an existing paralysis is of graver significance." He has also found that, as a general rule, the smaller the number of cells present in the cerebro-spinal fluid during the first twelve or eighteen hours from the onset of the illness, the less chance is there of paralysis occurring. In the fatal cases the number of cells is almost invariably very large—over 700 in the c.mm. After the first twenty-four to thirty-six hours the examination of the cerebro-spinal fluid has no prognostic value.

In the common sporadic cases of the disease, in which there is well-marked paralysis of one limb, the chance of complete recovery is small. We have only seen one or two instances. In the milder and more irregular cases met with during an epidemic, however, and especially in those in which several limbs or other parts are partially affected, the chances of recovery are much greater; and complete, or almost complete, recovery of power is quite common.

In cases in which the paralysis passes off entirely, it usually does so within a month or two of the onset of the attack. The younger the children the more likely are they to recover.

The proportion of recoveries has varied considerably in different epidemics. Wickman reported that in 530 cases which were re-examined from twelve to eighteen months after the attack, 56 per cent. showed paralysis, and 44 per cent. had recovered. In most epidemics, however, the proportion of complete recoveries is not nearly so large as this; in several it has been estimated as about 16 or 17 per cent. In those cases in which several limbs are involved, some of the parts affected generally recover entirely.

In any case, most of the progress takes place within the first six months; but, under careful treatment, improvement

may continue for as long as two years. If, after twelve months' treatment, the power shows no sign of returning, little improvement can be hoped for. If, however, an old-standing case which was not properly treated at first comes under energetic management, even several years later, considerable benefit is sometimes obtained. The prognosis as to the degree of recovery possible depends, of course, largely on the extent to which skilled treatment is carried out.

Treatment.—If the condition is recognised during the pre-paralytic stage then convalescent serum, *i.e.*, serum from a patient who has recovered from the disease, should be used. The dosage depends on the age of the patient, the degree of toxæmia, and the duration of the illness. An initial dose of 50 c.c. is recommended by Macnamara and Morgan,¹ as much as possible being introduced intrathecally and the remainder intravenously. If the dose has been sufficient the temperature falls and the general condition is improved. If there is no marked improvement within eighteen hours the dose has been insufficient, and a further 30 or 40 c.c. should be given intravenously. Serum therapy, as already mentioned, is of no value after the onset of paralysis.

Otherwise the case is treated as any other acute febrile illness—a bland fluid diet and nerve sedative (antipyrine or phenacetine) to relieve pain. When pain is very severe, morphia may be required. In some cases relief to pain is obtained by lumbar puncture.

From the first appearance of paralysis the posture of the affected limb must be arranged so that the affected muscles are kept in a state of relaxation and not damaged through overstretching by the opposing muscles, or by the force of gravity. When the deltoid is paralysed the arm must be raised from the side, and when the foot drops, owing to paralysis of the anterior tibial group, the ankle must be held at a right angle. The limbs may be kept in the position desired by encasing them in ordinary splints or by a light plaster. Such methods are quite suitable in the early stages, but as the limb will probably be required to be kept at rest for months, or possibly years, it is advisable to provide splints made of vulcanised fibre or celluloid or pexuloid, which can be moulded to fit accurately the limb or other part of the body.

¹ J. Macnamara and F. G. Morgan, *Lancet*, 1932, i., 469 and 527.

When the acute phase of the disease has subsided and all pain has disappeared it is wise to allow the patient up, but only if the paralysed muscles are adequately supported by proper splints. This may necessitate the limbs and trunk being fixed, and on account of their strength and lightness pexuloid splints are undoubtedly the best. These splints are now made at most Children's Hospitals or at Municipal Orthopædic Departments. The method of preparing such splints is described by Batten in his "Lumleian Lectures."¹

As the muscles show signs of recovery by slight voluntary contractions this should be encouraged, but care must be taken that there results no exhaustion from undue strain. It is necessary that at first the limb be entirely supported and any movements be aided and only allowed to overcome the slightest resistance. Severe passive movements must be avoided, as in this way the weakened muscles are stretched and the paralysis is kept up. Gradually the muscles are re-educated to perform stronger and stronger contractions. By placing the child in a warm bath gravity is to a great extent eliminated and the movements are performed with greater ease. For this re-education it is a great advantage if the services of a trained masseuse can be obtained, but the mother or nurse, if intelligent, can usually be taught to do it fairly well. The "Directions to Mothers regarding Children with Paralysed Limbs," in use at the Great Ormond Street Children's Hospital, which will be found in Appendix C, p. 1021, give plain instructions which are easily carried out by mothers under supervision.

The affected limb must be kept constantly warm by padded stockings and hot bottles; and its circulation should be encouraged by the use of alternate hot and cold douches.

Massage in poliomyelitis is of no value so far as helping the recovery of the muscle is concerned. Massage may, however, improve the tone of the skin and render it less susceptible to chilblains, which are often a troublesome feature of the late stage of the disease. Electricity, once so much practised in this disease, is also of no therapeutic value. Any current which will cause contractions of the weakened muscles is very painful and upsets the child.

The amount of recovery depends on the extent to which the motor cells have escaped destruction. If the cells are

¹ F. E. Batten, *Brain*, 1916, xxxix., 115.

destroyed nothing will bring about a cure. Often, however, many of the cells have simply been put out of action by pressure of the inflammatory exudate, and recover when the swelling subsides. All that really can be done is to keep the muscles in as good a state as possible. This we do, as described above, by putting the muscles which are paralysed in a state of relaxation. We know from experience that any healthy muscle can be paralysed by stretching, far less one which has been damaged.¹ Hence the splints must be worn night and day, and as long as any support is necessary. It may take two or three years, or even longer, for all recovery that will occur to take place.

This is not the place to deal with the surgical and orthopædic treatment of the deformities due to poliomyelitis. It may be well, however, to refer to the great practical value of the advances made in this subject during recent years. The measures employed consist in rectifying such deformities as contractures of the hip, knee, and other joints, the use of mechanical supports of various kinds, transplantation of tendons, the fixation of flail joints, nerve-crossing, and various other devices which make up an important and difficult part of the work of the modern orthopædic surgeon.²

Isolation.—It is very rare that direct infection from the patient occurs in this disease; but it is certainly possible. The other children should not therefore be allowed to go near him during the first three weeks, or even longer, if there is any nasal catarrh. During an epidemic, members of the household who may be carriers should be isolated to a reasonable extent for ten days. They should also be recommended to cleanse the nose and mouth frequently with a saline or alkaline lotion. This does just as well as an antiseptic solution, for the normal nasal secretions have a strong power of neutralising the poliomyelitis virus. Possible carriers must be careful always to wash their hands when they have been contaminated by secretions of the mouth or nose (as in coughing and sneezing), and after defæcation. Kissing must, of course, be forbidden.

¹ R. Jones, *Brit. Med. Journ.*, 1914, i., 1165.

² R. W. Lovett, *The Treatment of Infantile Paralysis*, London, (Heinemann), 1916.

Encephalitis Lethargica (*Epidemic Encephalitis*).

During the winter of 1916-17 there occurred in Vienna a considerable number of cases of an epidemic cerebral disease, the symptoms and pathological lesions of which showed it to be a clinical entity of an unfamiliar type.¹ In the early spring of 1918 similar cases began to appear in England²; and about the same time a more severe outbreak of the same disease was observed in France.³ Cases were also reported from America in the autumn of 1918; and since then the disease has become increasingly prevalent in this and other countries, and much has been written on the subject. The disease is now known as lethargic or epidemic encephalitis, and it has been shown that it is not an altogether new disease, but one which is known to have occurred in past times in various countries.

Etiology—*Seasonal Incidence*.—The disease may occur at any season, but it is most prevalent during the colder months, December to February.

Age and Sex Incidence.—Any age, from birth onwards, may be affected. The proportion of children varies considerably in different epidemics. In some, as in the first outbreak of the disease in Edinburgh, there have been very few cases under 20. In most, the number of children has varied from 20 to over 50 per cent. of the whole. The following Table shows the age incidence according to sex of cases which occurred in Glasgow during the years 1918-1924:—

Percentage Distribution in each Sex with Morbidity Rates at all Ages in Encephalitis Lethargica.

Years . .	0-	5-	10-	15-	25-	35-	45-	55-	65-	Morbidity Rate per cent. all ages.
Males . .	6	13	20	26	10	13	7	4	1	16
Females . .	5	12	11	36	15	9	9	3	0	27

(From A. K. Chalmers, *Glasg. Med. Journ.*, 1925, civ., N.S., 216.)

The sexes are usually stated to be equally affected. Of the above cases recorded by Chalmers, 375 were males and 242 females.

¹ Economo, *Wiener klin. Wochenschr.*, 10th May 1917, 581.
² S. A. Kinnier Wilson, *Lancet*, 1918, ii., 7.
³ A. Netter, *Soc. Méd. des Hôpitaux de Paris*, 22nd March, 12th, 19th, 26th April, and 3rd May 1918; *Bull. de l'Acad. de Méd.*, 7th May 1918, lxxix., 337; and *ibid.*, 1920, lxxxiii., 303.

Infection.—There can be little doubt that the disease spreads by personal contact; but the degree of contagiousness is not great; and it is interesting, in this connection, to note that doctors and nurses are very rarely affected. The occurrence of more than one case in a family is extremely rare; but one of us (J. T.) saw in the practice of his friend, Dr Goossens of Bruges, a family of three children suffering from it at the same time.

Pathology.—The bacteriology of the disease is still undetermined. The virus, which is filtrable, has been found in the mouth and nasopharynx, as well as in the tissues of the nervous system; and it probably passes from the throat to the mid-brain and medulla through the lymphatics.

The main pathological lesion consists in an acute diffuse encephalo-meningitis—the capillaries, veins, and arterioles being surrounded by numerous lymphocytes, as occurs in poliomyelitis; and small hæmorrhages are often found.

Symptoms.—The onset is almost invariably sudden. The child may go to school quite well and return home two hours later extremely ill, or he may retire to bed in his usual good health and awaken during the night violently delirious. In the majority of cases there is nothing characteristic in the early stages, and hence at first the true nature of the illness not infrequently passes unrecognised. There may simply be the features of a chill or a mild attack of influenza, as, *e.g.*, fever and drowsiness. In some cases there occur delirium, convulsions, paralysis, or chorea pointing to cerebral mischief.

There is probably a degree of *fever* in all cases. In all these observed at R.H.S.C., Glasgow, during the early stage fever was present. The fever is seldom higher than 103° F., and usually varies between 102° and 103° F., but in rapidly fatal cases temperatures of 106° and 107° may be registered.

Drowsiness is, next to fever, the most constant symptom, but it varies much in severity. It was present in 60 per cent. of the cases observed at R.H.S.C., Glasgow. The degree of drowsiness may be no more than is usually met with in febrile conditions during childhood, but in many instances it is profound and peculiarly characteristic. The child is constantly asleep and requires to be roused to be fed and to have the bowel and bladder attended to. When roused, however, he is as a rule quite *compos mentis*, recalling the picture of cerebral abscess.

In a certain proportion of the cases there occur, as mentioned above, symptoms pointing to cerebral involvement. A transient *diplopia* is particularly common. Foster Moore¹ found ocular paralysis in 75 per cent. of a series of 97 cases, and it was present in 74·5 per cent. of 40 acute examples of the condition under observation in R.H.S.C., Glasgow, but its transient nature (it seldom lasts longer than twelve hours) renders it liable to be overlooked. *Optic neuritis* is rare, but a mild degree of *retinitis* is not uncommon.

The symptomatology has varied in different epidemics, and in 1920 *chorea* was specially characteristic.² Many cases during that year were admitted to hospital with a diagnosis of acute chorea. The chorea appears suddenly and rapidly increases in severity, so that within a few hours the child is constantly twisting and turning, and it is with the greatest difficulty that he can be restrained from injuring himself.

When the disease first made its appearance it was frequently mistaken for tuberculous meningitis, but examination of the cerebro-spinal fluid failed to reveal the features present in that disease. The *cerebro-spinal fluid* is usually under increased pressure, but it is clear and presents no or only a slight increase in the protein content. During the acute phase of the disease there may be a slight increase in the number of cells, up to 30 per cubic millimetre, but a count of as many as 100 per cubic millimetre has been recorded. The most striking change in the cerebro-spinal fluid is the reaction to gold chloride (Lange's test).³ Of the cases observed at R.H.S.C., Glasgow, this test was positive in 29 of 39 examined during the acute phase, in 19 of 26 examined between two months and one year after the onset, and in 3 out of 9 examined between one year and three years after the commencement of the illness. The reaction, which is either of the luetic or paretic type, may be negative in the early stage and only become positive later on (p. 777).

After persisting for a variable time—usually days, but occasionally weeks—the fever, drowsiness, and chorea disappear, and the child seems as if he is making a complete recovery. Soon, however, a new phase appears with the development of one of the most troublesome and most prominent features

¹ R. F. Moore, *Medical Ophthalmology*, London, 1922, 112.

² L. Findlay and C. Shiskin, *Glasg. Med. Journ.*, 1921, xcv., 18.

³ *Ibid.*

of the disease as it affects the child. It may have been that the early manifestations were mild and uncharacteristic, and that their true nature was not appreciated, so that the patient first comes under observation on account of this phenomenon, viz., the *disturbance of the sleep rhythm*.^{1,2} The sleep sequence is reversed. The child is drowsy during the day and wakeful by night. On going to bed at night the child cannot fall asleep but tosses and turns, rearranges the blankets, now tries the pillow at the head of the bed and now at the foot, gets up and walks about, or reads or plays games until about 3 or 4 o'clock in the morning, when he ultimately falls asleep. Sleep is then deep and he cannot be roused at the usual time and may remain in bed till noon. During the day he remains dull and drowsy and tends to fall asleep whenever left at rest. But as the day wears on he becomes less sleepy and in the evening is comparatively bright.

About the same time *psychic disturbances* with mental and moral deterioration may become apparent. Hyperpnœa, constant or periodic, sialorrhœa with the habit of spitting, a spasmodic cough or peculiar grunt, myoclonic contractions of various groups of muscles with or without hiccough, depending on whether the thoracic or abdominal muscles are implicated, are not infrequent. As a result of the hyperpnœa, alkalosis with tetany may develop (pp. 547 and 568).

Often the child's disposition becomes changed. He is mischievous, disobedient, cruel, and deceitful, is subject to stealing, and, rarely, precocious sexually. Some mental deterioration is almost constant. Prior to the appearance of epidemic encephalitis, acquired mental deficiency was comparatively rare, and in most instances was due to lues, but during the last decade mental deficiency developing in later childhood has become much more frequent, and is due entirely to the ravages of this disease. Dawson and Conn³ have made a very complete study of the cases which passed through the R.H.S.C., Glasgow, and they found that the average intelligence of the encephalitic patients was definitely below the average of the non-encephalitic patients, as well as below that of their healthy brothers and

¹ L. Findlay and C. Shiskin, *Glasg. Med. Journ.*, 1921, xcv., 18.

² G. H. Anderson, *Quart. Journ. Med.*, 1923, xvi., 173.

³ S. Dawson and J. C. M. Conn, "Intelligence and Disease," *M.R.C. Report*, No. 162, 1931, 27; *Arch. Dis. Child.*, 1926, i., 357.

sisters. The average mental ratio of the encephalitics was 84.63, whereas that of the two other groups was 90. As a rule the mental deficiency is due to an arrest of development, and it is for this reason that the consequences of the disease are more serious in the younger and least developed children. At times, however, there is definite deterioration, as is evidenced from repeated mental tests. Usually, however, there is some improvement, although this may not be commensurate with the increase in age, and hence the child's mental ratio steadily falls.

A large proportion of the children ultimately develop the *Parkinsonian syndrome*. This sequela was noted in 30 per cent. of the cases observed at the R.H.S.C., Glasgow. It may set in very soon after the acute stage of the disease has passed off, or it may be delayed for as long as three years. The condition would appear to be relatively more frequent in boys than in girls, and in the older children than in the very young. All the features of the condition do not occur in every case, but facial immobility, a slurring speech of high pitch, and a stiff wooden gait are specially characteristic. A certain number present festination and retropulsion and severe tremor of the limbs may also be present. Many of the patients become absolutely bed-ridden. The interesting condition of "kinesia paradoxa," in which an apparently immobile patient suddenly presents sudden activity, is occasionally observed.

Diagnosis.—The diagnosis of epidemic encephalitis is often extremely difficult. This is specially true during the early stages, when the characteristic symptoms—diplopia, chorea, or profound lethargy—are not present. If there is an epidemic prevalent one can found a diagnosis on a more slender basis than when the example is sporadic, but often a diagnosis cannot be arrived at until the disease has unfolded itself by the development of the so-called sequelæ, viz., night restlessness, mental and moral changes, or the Parkinsonian syndrome.

In the early stages the characteristic features are diplopia, which is usually transient, chorea of sudden onset and rapidly increasing severity, very profound lethargy without real dulling of the sensorium, and a cerebro-spinal fluid under increased pressure, with a very moderate increase in lymphocytes, a negative Pandy's test showing no increase in its protein content, and a positive Lange's reaction either of the luetic or paretic type. The milder examples are likely to be mistaken

for a chill, pneumonia, or influenza, and the more severe varieties for tuberculous meningitis, cerebral tumour or abscess.

As previously mentioned, many of the examples first come under observation on account of the sequelæ. If it is remembered that these phenomena are usually due to this infection, careful inquiry will elicit the history of some acute illness, months or perhaps years previously, since when the child has never really been well and has passed through the various phases of the disease as described under symptomatology. Even at this late date the cerebro-spinal fluid may give a positive Lange reaction, but there will be no increase in cells, the protein content will be normal and Wassermann's test negative, all of which differentiate it sharply from neuro-syphilis.

Prognosis.—There seems to be considerable difference of opinion regarding the prognosis as to life in this disease in the case of children. In A. C. Parson's statistics,¹ more than two-thirds of the cases under five died, and nearly a half of those between five and ten years old. In our experience the outlook so far as life is concerned has not been so serious. Of the cases observed at the R.H.S.C., Glasgow, 10 per cent. died during the acute stage within the first sixteen days. A high temperature and severe chorea were grave symptoms. Of the children who got over the acute stage, 10 per cent. died at a later period. Neither sex nor age seemed to be factors influencing the mortality.

Prognosis regarding ultimate recovery is, however, exceedingly grave. Of the sixty-five children who survived after being under observation in R.H.S.C., Glasgow, only three, *i.e.* 4.6 per cent., became absolutely normal physically and mentally. The others are all mentally or morally affected, so that they require to be confined in an institution, or are such severe examples of Parkinsonism that they are either confined to the house or are bed-ridden.² Robb³ only reports 5 per cent. of recoveries, Nonne⁴ only very few in 161, whereas Shrubsall⁵ found the recovery rate as high as 23.4 per cent.

¹ A. C. Parson, *Ministry of Health Report on Enceph. Leth.*, London, 1922, 114.

² M. M. Stevenson, *Arch. Dis. Child.*, 1928, iii., 57.

³ A. G. Robb, *Brit. Med. Journ.*, 1925, ii., 644.

⁴ N. Nonne, "Cong. Int. Med.," *Lancet*, 1923, i., 866.

⁵ P. C. Shrubsall, *Proc. Roy. Soc. Med.*, 1925, xviii. (Sect. Neur., 21).

Treatment —In the acute stage nothing can be done apart from securing rest and quiet and the treatment of symptoms. In cases of insomnia little good results from giving hypnotics even in very large doses. Temporary benefit has sometimes followed a rise of temperature produced by the subcutaneous injection of milk, and the use of saline infusions is occasionally helpful.

In the ordinarily severe chronic cases a change to new and quieter surroundings, with judicious care and discipline, is often followed by distinct improvement. Removal to an Institution for the Mentally Defective is often necessary and usually beneficial. No medicinal treatment would seem to have any permanent beneficial effect on any phase of the disease.

CHAPTER XXXI

MENINGITIS AND HYDROCEPHALUS

MENINGITIS may have many causes. During childhood the tubercle bacillus, the *Spirochæta pallida*, the meningococcus, the pneumococcus, the streptococcus, and the influenza bacillus are the usual etiological organisms. In the new-born the *Bacillus coli* is a frequent cause, and at times the gonococcus has been the exciting agent. Of 789 examples of meningitis observed in the R.H.S.C., Glasgow, during the course of the years 1914 to 1929 inclusive, the relative frequency of the various types is shown in the following Table:—

Frequency of Various Types of Meningitis.

Type.	No.
Tuberculous meningitis	463
Meningococcal „	173
Pneumococcal „	77
Influenzal „	20
Streptococcal „	12
Syphilitic „	5
<i>Bacillus coli</i> „	1
Serous „	38
Total	789

These figures can only be taken to give a general idea of the relative incidence of the various types of meningitis, and probably underestimate the frequency of meningitis due to the meningococcus. The comparatively low proportion of the examples of this type of the disease in the above series is due in part to the fact that no real epidemic occurred during the period under observation, and in part because the admission of cases of cerebro-spinal fever was limited. A more correct idea of the relative incidence of this disease is revealed in the notifications of the city of Glasgow as a whole. In 1930 there were notified 214 examples of tuberculous meningitis, and 148 cases of cerebro-spinal fever.

The incidence of streptococcal meningitis is less than most

authors record, and is probably to be accounted for by the fact that these statistics are entirely compiled from a medical department.

Although there follows, in conformity with the usual custom, separate accounts of the main types of meningitis, it must be appreciated that the symptomatology of the disease is alike, no matter what the causal factor, and that it is only by the discovery of the etiological organism in the cerebro-spinal fluid that the particular variety of the disease can be recognised. The cellular deposit is of no help, as the nature of the predominant cell is influenced by the stage of the illness as much as by the nature of the organism. While in tuberculous meningitis lymphocytes predominate in the early stages and polymorphs towards the end, in meningococcal meningitis the polymorphonuclear exudate of the early days becomes replaced by a lymphocytic picture as the inflammation subsides. This is well shown in the following examples:—

Table showing Type of Cell predominant in C. S. F. in Tuberculous and Meningococcal Meningitis at different stages of the disease.

Day of Disease.	Tuberculous Meningitis.		Meningococcal Meningitis.	
	Lymphocytes.	Polymorphs.	Lymphocytes.	Polymorphs.
	Per cent.	Per cent.	Per cent.	Per cent.
3rd	13	87 *
5th . .	99	1	20	80 *
7th	24	76
13th . .	94	6	90	10 *
14th	70	30
16th . .	60	40
22nd . .	40	60	96	4
29th	100	0

* Meningococci present in films.

It is equally important to bear in mind that it is only by an examination of the cerebro-spinal fluid, and the detection of evident inflammatory products, that the presence of meningitis can be definitely diagnosed, because so often do general toxic conditions present meningeal symptoms (meningismus).

The Table on pp. 776-777 gives the details of the changes in the cerebro-spinal fluid observed in the different diseases of the central nervous system.

Table showing the Changes in

Condition.	Pressure.	Appearance.	Cytology.		Bacteri- ology.
			Number per cub. mm.	Type.	
Health . . .	Normal	Crystal clear	Less than 5	Lymphocytes	Sterile
Tuberculous Meningitis	Increased	Not just clear or opalescent	50 to 500	Lymphocytes in early stage and polymorphs in late stage	Tubercle bacilli
Meningococcus Meningitis	"	Turbid	2000 or more	Polymorphs early and lymphocytes late	Meningo- cocci
Suppurative Meningitis	"	"	"	Polymorphs	Particular etiological organism
Poliomyelitis or Polioencephalitis	"	Opalescent	30 to 2000	Polymorphs 90 per cent. early, lymphocytes 100 per cent. late	Sterile
Lethargic or Epidemic En- cephalitis	"	Not just crystal clear	5 " 50	Lymphocytes	"
Meningo- Encephalitis (luetic)	"	Not just clear	5 " 50	"	"
Cerebral Abscess	"	Clear	150 - 2000 +	Polymorphs	"
Cerebral Tumour	"	Clear or slightly xantho- chromatic	Normal	...	"
Froin's Syn- drome (Spinal Pressure)	Normal or diminished	Yellow and coagulates solid	Normal or slightly increased	Mono- nuclears	"

C. S. F. in Different Conditions.

Protein.		Glucose. Mgrm. per cent.	Chlorides. Mgrm. per cent.	Gold Chloride. Lange Test.	W. R.
Mgrm. per cent.	Pandy's Test.				
0.02 to 0.04	Negative	0.05 to 0.06	0.72 to 0.75	0,000,000,000	Negative
0.05 „ 0.2	Positive	? diminished	0.58 „ 0.68	0,000,235,532	„
0.15 „ 0.75	Strongly positive	diminished	0.58 „ 0.63	0,000,235,532	„
0.15 „ 0.75	Strongly positive	„	0.58 „ 0.63	0,000,235,532	„
0.05 „ 0.2 Higher read- ings later	Positive	0.05 to 0.06	0.72 „ 0.75	0,000,123,210	„
Normal or slightly increased	„	?	Normal	5,554,310,000 1,344,210,000	„
0.05 to 0.25	„	?	„	5,554,310,000	Positive
0.05 „ 0.15	„	?	„	?	Negative
0.02 „ 0.1	May be positive	?	„	0,000,000,000	„
0.5 „ 4.0	Strongly positive	Normal	„	0,000,000,000	„

Tuberculous Meningitis.

Tuberculous meningitis is a very common disease of childhood, especially among the lower classes. It is most frequently met with during the second and third years. It may occur, however, as early as the third month or even sooner; and it is not uncommon at any later period of childhood.

In many cases there is no family history of tuberculous disease and no clear indication of where the infection has come from. The milk supply, however, is often under suspicion, and, not uncommonly, we find that a phthisical relative or nurse has lived in the same rooms or been much with the child.

The meningeal affection is probably always secondary to a tuberculous focus elsewhere. This may be in the bronchial or mesenteric glands, in the bones or joints, the lungs, the brain, or the ears. But, during life, there is frequently no indication as to where it is situated. In some cases there is an account of a head injury. In a considerable proportion of the cases there is a history of the child's resisting power having been recently lowered by an attack of measles or whooping-cough.

Symptoms.—Children who have been debilitated by gross tuberculous lesions are liable to be suddenly attacked by severe symptoms of tuberculous meningitis. The patient, who has been in his usual languid state of health, suddenly develops paralysis of one or more limbs or of the face or ocular muscles, and soon after has a convulsion or becomes very drowsy. Such cases generally have a rapidly fatal course.

In the great majority of cases, however, the initial lesion is one which has not hitherto materially affected the child's general health. When this is so, the onset of the brain symptoms is generally very insidious.

In a typical case the onset of characteristic symptoms is preceded by a *premonitory stage*, which may last for two or three weeks, or even for two or three months. During this period the child is vaguely out of sorts. He is languid and irritable during the day, and restless and wakeful at nights. He loses appetite and weight, and sometimes vomits with no apparent cause. He is constipated, has occasional rises of temperature, is drowsy at times, and sometimes flushes and complains of headache. Occasionally incontinence of urine or fæces may begin. The pulse may be slow and irregular.

If he is old enough to be speaking, he will probably stop doing so. In many ways his mother finds him changed and unlike himself in disposition.

At this period, while it is obvious that the child is far from well, there are usually no symptoms present which might not be attributed to dyspeptic derangement in a teething baby. In older children and adolescents the early symptoms of tuberculous meningitis may closely resemble those of hysteria.

Sooner or later the disease shows itself more distinctly by the marked aggravation of some of the already existing equivocal symptoms and the occurrence of others of a more definitely cerebral character. The *period of invasion* may now be held to have begun. It is customary to divide this period into three not very well-defined stages. The first is one of *irritation*, and corresponds to the implication of the meninges and cortex. The second is that of *pressure*, during the accumulation of fluid in the ventricles. The last is characterised by *relaxation* and paralysis, and indicates the involvement of the medulla.

Tuberculous meningitis is such a treacherous disease that we can hardly speak of any group of its symptoms as pathognomonic. The most characteristic combination, however, is that of *vomiting*, *headache*, and *drowsiness* with *constipation*. This should always arouse suspicion, and the onset of *convulsions*, in addition, may be held as confirmatory.

Early in this stage the peculiar *facies* which has been already described (Figs. 4, 6, and 7, p. 9) generally develops; the cheeks are flushed, and the *tache cérébrale* is usually present. The patient tends to lie constantly on his side with the limbs fully flexed. He is very irritable, and resents being disturbed. If the bed-clothes are pulled off, he clutches at them, draws them back and screams irritably (*Stocker's sign*). He may show marked *photophobia*, but this is not so common a symptom in children as in older patients. Occasionally the child may utter a sudden sharp scream, the so-called *hydrocephalic cry*. Far too much prominence has been given to this symptom, which is not an important one, and occurs only in a small minority of the cases. A piercing scream is much more characteristic of acute ear disease than of meningitis.

The *headache* is sometimes very severe, and a bad headache in a child under five years of age is always an anxious

symptom, as it is rarely in them due to functional disease. Occasionally severe pain is complained of in some other part of the body. If the *drowsiness* is marked it also is very significant, provided the action of alcohol and other narcotics can be excluded. *Vomiting* is nearly always present at some period in the case. It may last long, but sometimes it only occurs two or three times at the beginning. *Constipation* is also a fairly constant symptom; rarely there is diarrhoea. Unless abdominal tuberculosis is present, there is usually, though not always, marked *retraction of the abdomen* (Fig. 236).

At first the *pulse* may be regular and simply accelerated, but soon it develops the peculiar characters already described

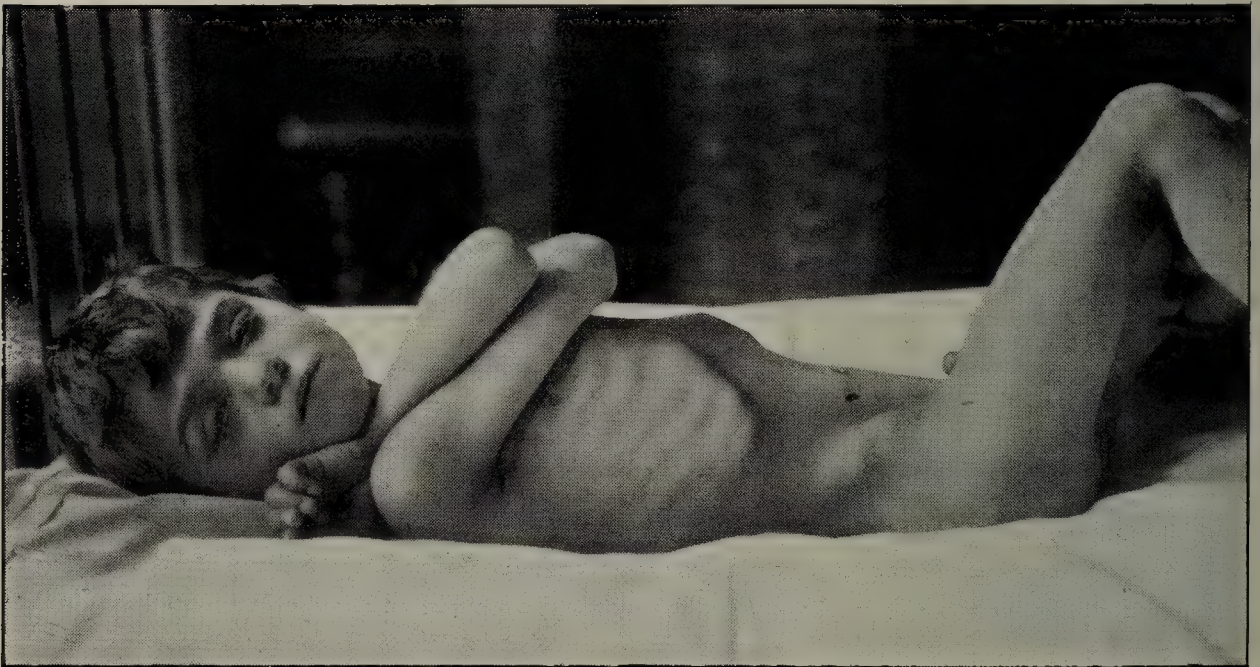


FIG. 236.—Tuberculous Meningitis with Retracted Abdomen.

(p. 574). The *respiration* early becomes irregular, and frequent deep sighing is very characteristic. The fontanelle is tense and pulsating, the neck stiff at times, and occasionally distinctly retracted. *Head retraction*, however, is rarely very marked in this form of meningitis, nor is it often continued for any length of time. Kernig's sign occurs at one time or another in about half the cases, and Babinski's less frequently.

Convulsions in the early stage are less characteristic of this than of some other forms of meningitis, but they are not uncommon, and may occur at any period of the disease. They may be followed by paralysis of limbs and by rigidity. The pupils are usually contracted and sometimes oscillate, and there are often irregular nystagmoid movements of the eyeballs. Various degrees of *squinting* are common, and ptosis is also

often met with. The *temperature* is irregular, though rarely very high. Occasionally it remains normal or subnormal until during the last few days, when it usually rises: on rare occasions the temperature falls instead of rises. The blood often shows a considerable leucocytosis, greater than in the other forms of tuberculous disease.

On lumbar puncture the fluid generally spurts out owing to the increased pressure. It is very slightly turbid, contains an increased number of cells (50 to 500), which, in the early stage of the disease, are lymphocytic in nature, but which later on may be entirely polymorphonuclear in type. There is a moderate increase in the globulin, with a diminution in both the sugar and chlorides. Tubercle bacilli can usually be found, but they are as a rule scanty and require long search. The best method for finding bacilli is to allow the fluid to stand till a coagulum (so-called pellicle) forms. The fluid with the pellicle is poured into a watch-glass and the pellicle floated on to a cigarette-paper, then transferred to a clean microscopic slide, on which it is fixed by rubbing until dry with a piece of blotting-paper. On removing the cigarette-paper, the pellicle will be found uniformly spread and most suitable for microscopic examination.¹ Some authors² have recommended cistern puncture (p. 690) in suspected cases as they hold that bacilli are more numerous at this site; in our experience, no better results are obtained by cistern puncture than by lumbar puncture.

In the *second or pressure stage* of the illness drowsiness is the most marked feature. The vomiting usually ceases, the pulse gets more markedly irregular and slow, various local paralyses may occur, and quasi-purposive movements of the face, lips, jaws, and hands are common. Grinding of the teeth is frequently heard in older children. The urine and fæces often pass involuntarily. Cheyne-Stokes' respiration develops, and local or general convulsions may occur.

Finally, the *stage of paralysis and coma* sets in. In it the child lies on his back, more or less entirely unconscious, with his limbs extended and his hands often crossing one another over the pubes. The pupils are widely dilated, the conjunctivæ insensitive and often covered with shreds of muco-pus. Generally a degree of optic neuritis is present, and sometimes choroidal

¹ This technique was devised by Mr Nathaniel Smith, *Arch. Dis. Child.*, 1930, v., 187.

² D. Stewart, *Edin. Med. Journ.*, 1927, N.S., xxxiv., 36.

tubercles are found ; but these are more characteristic of general tuberculosis. During this stage the bulging fontanelle often becomes flattened, the pulse is regular, very rapid, and weak, the respiration shallow and quick, and the temperature irregular. Swallowing becomes increasingly difficult. In the course of the last week, pyuria (from *Bacillus coli* infection) is common ; and glycosuria is also often met with, as Still and Garrod have pointed out (p. 527).

The final stage may be painfully prolonged for many days. Sometimes there is apparent improvement for a day or two before the end.

The **duration** of the illness varies considerably in different cases. In cachectic children, as already mentioned, death may occur within a few days, and generally within a week. In ordinarily vigorous infants the symptoms may be expected to last about three weeks before death takes place. Some cases go on much longer, however, when carefully nursed, and Barlow mentions one which lasted for sixty-three days.

Diagnosis.—Owing to the number of its symptoms and their indefinite character, tuberculous meningitis is often mistaken for other conditions at different stages of its course. Examination of the cerebro-spinal fluid is, as already mentioned, our only certain means of arriving at a diagnosis and should never be omitted in any suspicious case.

Dyspeptic vomiting, especially in teething children, sometimes gives rise to difficulty. And its symptoms may be so like those of early tuberculous meningitis that the diagnosis is impossible in the early stages. In judging between the two conditions we should estimate carefully the other symptoms of digestive disturbance and the state of the gums. We should also look out for retraction of the abdomen, slowness and irregularity of the pulse, and a sluggish reaction of the pupils. The occurrence of convulsions is in favour of meningitis, although it may also occur in the other disease. A dose of castor oil will sometimes clear up perplexing symptoms in a dyspeptic and teething child.

In the cases in which *cyclic vomiting* begins in young infants it may simulate tuberculous meningitis to a considerable extent, as may *gastric influenza*, and also the onset of various of the *exanthemata* in nervous children.

Cases of *typhoid fever* occasionally resemble tuberculous meningitis very closely. In considering the differential

diagnosis we must note the temperature, which is usually higher in typhoid, and the pulse, which is less slow and more regular. The typhoid patient tends to lie on his back, is not usually irritable, and does not show Stocker's sign. He also shows no cervical rigidity, the abdomen is more or less full instead of being retracted, and the spleen is usually enlarged. After the eighth day spots may be found, and a blood culture or Widal's test will generally decide the matter.

The extreme irritability, change in character, and constipation which occur in the early stage of *erythrædema* often give rise to a suspicion that tuberculous meningitis may be beginning.

Acute pneumonia, especially when it affects the apex, also gives rise to mistakes. The rapid onset with high fever, the pneumonic facies (p. 7), the regular pulse, and the early appearance of rapid respiration, should enable the diagnosis to be readily made by anyone who remembers the occurrence of "cerebral pneumonia." A high degree of polymorphonuclear leucocytosis is in favour of pneumonia.

Acute middle-ear disease is another condition which simulates meningitis. It sometimes gives rise to irregular pyrexia, irritability, drowsiness, screaming, vomiting, some bulging of the fontanelle and stiffness of the neck, and to convulsions. The temperature is generally higher and more continuous than in tuberculous meningitis, the abdomen is not retracted, and there is deafness. An examination of the ears or the appearance of discharge from the meatus settles the question.

Uræmia and *pyelonephritis* in young children occasionally present symptoms difficult to distinguish from those of tuberculous meningitis, but the condition of the urine soon clears up the diagnosis.

Sub-acute liver atrophy also at times gives rise to symptoms which are very like those of tuberculous meningitis.

Cases of *spurious hydrocephalus* are to be distinguished by the history of recent severe diarrhœa and by the presence of a depressed fontanelle, frequently also by a distended abdomen.

Cases of *polioencephalitis*, as also poliomyelitis, often closely resemble tuberculous meningitis in the early stages, and indeed, unless in the difference in the chloride contents of the cerebrospinal fluids, their differentiation may be impossible (see Table, p. 776). In poliomyelitis, however, there is seldom the dulling of the sensorium so characteristic of meningitis.

The diagnosis between tuberculous meningitis and *intra-cranial tumour* has been already referred to (p. 751). We must bear in mind the rarity of localising symptoms from the latter in children under two.

Treatment.—When tuberculous meningitis is really present, treatment is probably never of any use. It is almost always well, however, to order some treatment, partly for the parents' sake and partly because we cannot be absolutely certain, unless from the presence of tubercle bacilli in the cerebro-spinal fluid, that our diagnosis is correct.

The favourite remedy to administer in these cases is mercury, and it may be prescribed internally, in the form of liq. hydrarg. perchlor. (1 drm.) or as calomel (which has the further advantage of counteracting the constipation), or it may be applied externally as mercurial ointment. Small doses of pot. iodid. may also be given, or iodoform ointment (10 per cent.) may be applied to the head.

When severe headache is present, lumbar puncture or the application of one or two leeches to the temple sometimes gives relief; and chloral may be given if convulsions set in. Surgical treatment has been attempted from time to time, but it cannot be said ever to have been proved to be of value. Lumbar puncture certainly gives temporary relief in a few cases. In the great majority, however, it has no effect at all; and it has the disadvantage of sometimes prolonging the hopeless illness considerably, although in other cases it seems to hasten the end.

Meningococcus Meningitis

(Epidemic Meningitis—Cerebro-spinal Fever).

Next to tuberculous meningitis this is the variety most frequently met with during infancy and childhood. Meningococcus meningitis is always present as a sporadic disease, especially during infancy. Of 154 examples observed in R.H.S.C., Glasgow, between 1914 and 1929 (non-epidemic years), 94, or 61 per cent., were infants under one year of age.

Epidemics of varying severity occur from time to time. The last serious outbreak in Glasgow occurred during the winter of 1906 to 1907. During that epidemic the proportion of cases under one year diminished, but still 53 per cent. of the patients were children under five years of age.

Although the disease is definitely infectious, seldom does more than one case occur in a household. During epidemic times, however, multiple cases in a household increase in frequency.



FIG. 237.—Epidemic Cerebro-spinal Meningitis. (Dr. J. S. Fowler's case.)

Symptoms.—The patient has usually, hitherto, been a strong healthy child, and the onset is almost always very sudden. There is high fever, severe headache, and pains in the nape of the neck, the back, sides, and elsewhere. Vomiting almost always occurs,



FIG. 238.—Epidemic Cerebro-spinal Meningitis. (Dr. J. S. Fowler's case.)

often there is delirium, and frequently convulsions. The child looks and evidently feels very ill in most cases. Quite often there is difficulty in breathing and an increased rate of respiration. Severe abdominal pain is not uncommon.

Probably the most distinctive physical sign is the cervical rigidity. It is only absent when the patient is comatose, but at

first it may not be severe in degree. There is generally acute pain on the slightest attempt to bend the head forward. More or less marked head retraction soon sets in (Fig. 237), and in the later stages the whole body is stiff and extreme opisthotonos is common (Fig. 238).

Kernig's sign is nearly always present; the knee-jerks may be normal or absent. In the early stages the abdominal reflexes are usually quite abolished (Fowler); but they return as the acute symptoms subside. Generally the patients are hyperæsthetic and very irritable if disturbed. So long as they are left alone, however, they are usually apathetic and show no signs of suffering. In the less acute cases the child's consciousness is often strangely little affected. In some cases there is rhinitis, and in a few purulent iritis.

Various rashes are met with. Herpes on the lips and elsewhere, like that in pneumonia, is fairly often present; and occasionally a mottled erythematous rash appears on the trunk and limbs. The most characteristic skin lesion, however, is the purpuric eruption from which the popular term "spotted fever" is derived. This rash was seen in about a quarter of the cases in the Edinburgh epidemic of 1907. Its presence is of bad omen and usually indicates a severe type of the disease. The temperature curve is very irregular.

In a few cases enlargement of the larger joints and swellings in the subcutaneous tissues of the limbs are seen. The blood shows a marked leucocytosis (10,000 to 45,000). Blindness, usually temporary, is often found at some stage of the case. Deafness may also be present.

Sometimes the onset is less acute but the symptoms are essentially the same. Head retraction (Fig. 239), convulsions and vomiting in these circumstances are particularly marked, but there is, at least at first, seldom the dulling of the sensorium present in the acuter varieties of the disease. Owing to the tendency for the exudate to be limited or most abundant at the base of the brain, occlusion of the foramen of Magendie is liable to occur, with consequent hydrocephalus and blindness. For this reason this type of the mischief is often spoken of as *posterior basic meningitis*. It is, however, like the other varieties of the disease, due to the meningococcus and is merely a subacute or chronic form of the disease. Lumbar puncture reveals the same picture as in the acute cases, except that the

organisms are particularly scanty and difficult to observe in film or isolate by culture. The duration of the illness is long, perhaps months, and there is usually great emaciation. Many acute cases pass into this stage and most of them terminate fatally.

Diagnosis.—The diagnosis depends on the finding of the meningococcus in the cerebro-spinal fluid. In the very early stage of the disease, and especially during epidemics, the cerebro-spinal fluid may be only very slightly turbid and the



FIG. 239.—Posterior Basic Meningitis. Extreme Opisthotonos.

organism only recovered by culture. As a rule, however, the fluid is definitely turbid and meningococci are easily recognised in films. On occasion the fluid may have the appearance of thick pus and can only be abstracted by suction.

It may not be possible to obtain fluid by lumbar puncture, and in these circumstances cistern puncture is advisable. If, however, it happens that the exudate blocks the foramen of Magendie, the cerebro-spinal fluid obtained either by lumbar or cistern puncture may fail to reveal organisms or indeed any evidence of meningeal reaction. This state of matters necessitates *ventricular puncture*, which is easily done so long as the anterior fontanelle is patent. The needle is introduced

at one of the lateral angles of the fontanelle and directed downwards, forwards, and slightly outwards towards the external canthus of the eye on the same side to reach the anterior horn of the ventricle, or downwards and backwards towards the mastoid process to explore the posterior horn. If there is any degree of hydrocephalus, which is almost certainly present under such conditions, this operation is one of the easiest, but if the ventricles are not dilated it may be one of extreme difficulty.

In the most acute, or "fulminant" cases, which are generally fatal in a few hours or days, the diagnosis may be difficult until lumbar puncture is done, because the severe general toxic symptoms more or less completely mask the special features of meningeal disease, and lumbar puncture may not be suggested.

In some cases of epidemic cerebro-spinal meningitis the disease sets in with severe diarrhoea and vomiting; and it is important to remember that several instances have been observed in which one child in a family has died of exceedingly acute gastro-enteritis with a purpuric rash but with no meningitis, while another has, soon after, had a typical attack of acute meningococcal meningitis. Occasionally the disease may commence with a multiple arthritis; exploration of the joints will reveal pus with meningococci.

In any child with a purpuric or petechial eruption, the question of cerebro-spinal fever should always be considered.

There is seldom any difficulty in distinguishing tuberculous meningitis from this disease. A period of indefinite illness or malaise preceding the apparent onset of the meningeal mischief points to the tuberculous infection. On occasion, however, tuberculous meningitis is quite sudden in onset. The form of meningitis which most closely resembles the epidemic cerebro-spinal variety in its symptoms is that produced by the influenza bacillus. Of all the varieties of suppurative meningitis it alone is the one which has a course of more than a week, and in this way simulates cerebro-spinal fever.

Prognosis.—Before the use of serum treatment the death-rate in this disease was very high, probably never less than 50 per cent., and often 70 to 80 per cent. or more. But even since the introduction of serum therapy, and especially in the case of the disease as it occurs sporadically, the death-rate

remains very high. One striking feature is the variation in the mortality rate from year to year. Of 59 cases treated in the City of Glasgow Fever Hospitals during 1927 death occurred in 83 per cent., while of 111 examples treated during 1930 a fatal issue resulted in only 61 per cent. This same variation in the annual mortality rate was observed in the case of the patients admitted to R.H.S.C., Glasgow, as the following Table shows:—

Mortality Rate in Cerebro-spinal Fever (R.H.S.C., Glasgow).

Year.	No.	Percentage Mortality.
1921 . . .	5	20
1922 . . .	5	60
1923 . . .	2	0
1924 . . .	12	50
1925 . . .	13	61
1926 . . .	17	70
1927 . . .	11	90
1928 . . .	17	59
1929 . . .	10	80
	—	—
Total . . .	92	63

That this behaviour of the death-rate is not peculiar to the West of Scotland is shown by the following results recorded by Du Bois and Neal of New York¹:—

Mortality Rate in Cerebro-spinal Fever (New York).

Year.	No.	Percentage Mortality.
1910-11 . . .	17	41
1911-12 . . .	25	60
1912-13 . . .	29	41
1913-14 . . .	41	26

The age of the patient would seem to influence the chance of recovery. The following Table shows the mortality rate according to age in the cases observed in R.H.S.C., Glasgow:—

Mortality Rate in Cerebro-spinal Fever according to Age (R.H.S.C., Glasgow).

Age.	Percentage Mortality.
Under 1 year . . .	83
1 to 5 years . . .	50
5 to 12 years . . .	37

¹ P. L. Du Bois and J. B. Neal, *Amer. Journ. Dis. Child.*, 1915, ix., 1.

Still another factor of importance influencing prognosis is the stage at which the disease is recognised and specific therapy instituted, as the following facts quoted by Holt¹ reveal:—

Mortality Rate according to Date of commencing Serum Treatment.

Time of injection—	Flexner (all sources, chiefly U.S.A.) Per cent.	Netter (France) Per cent.	Dopter (France) Per cent.
1st to 3rd day . . .	14.9	7.14	8.2
4th to 7th day . . .	22.0	11.1	14.4
After the 7th day . . .	36.4	23.5	24.1

It is generally stated that although life may be spared in a certain number of cases a complete recovery is rare. Hydrocephalus, mental deficiency, blindness, and deafness are looked upon as common sequelæ. Holt² writes that “in fully twenty-five per cent. serious sequelæ remain.” In our opinion the probability of there resulting any grave sequela in cerebro-spinal fever is exaggerated. If the child does not die, in our experience, he has generally made a complete recovery. In the case of twenty-six children, for the most part under one year of age, who had been under the care of one of us (L. F.) and recovered and were discharged from hospital, information was sought regarding their subsequent development five to eleven years later. Only three of the children could not be traced. Five had died in the interval, one from scarlet fever, one from measles, one from pneumococcal meningitis, one from convulsions, and one from some unknown cause. In the case of four children information was obtained from the parents or the family doctor that the children were in perfect health except for two who were slightly deaf, due in one instance to otitis media. Dr G. B. Fleming kindly saw and examined the remaining thirteen patients, and he reported that all were perfectly well and presented no disability excepting for one boy of nine and a half years who was somewhat dull, but quite definitely not a mental defective.

A similar absence of sequelæ is recorded by Wodarz.³ Of sixty-three children under ten years of age attacked during an epidemic in 1906, forty recovered. In 1929 he was able to trace nineteen, and all were absolutely normal except two,

¹ L. E. Holt, *Diseases of Infancy and Childhood*, New York, 1922, 716.

² *Ibid.*, p. 711.

³ A. Wodarz, *Zentralb. f. inn. Med.*, 1929, 1, 17.

who had slight deafness of one ear, but not of sufficient degree to interfere with ordinary work.

M'Lean and Caffey,¹ on the other hand, found that of forty-four children seen one to ten years after apparent recovery from meningococcus meningitis, which they had suffered from at ages varying between thirty-five days and four and a half years, some disability was presented by nine. Four were deaf-mutes, two had defective sight, two had hydrocephalus, and two were mentally defective. Five of the children had died in the interval and thirty were completely well. But Du Bois and Neal,² also writing from New York, only noted as sequelæ blindness in one child and deafness in two out of 112 children.

Treatment.—This consists in soothing the patient and sustaining his strength; and especially in the injection, into the spinal canal, of anti-meningococcus serum.

(1) The child must be kept very warm in bed and frequent hot baths should be given. These are of value in relieving the headache and restlessness, and in saving the strength. If the pains are severe, a small hypodermic of morphine may be required. In chronic cases the formation of bed-sores must be guarded against. No special dieting is usually necessary, but if there is much opisthotonos tube-feeding may be required for a time to ensure the child's getting enough food, and to obviate the risk of inhalation-pneumonia.

(2) The use of anti-meningococcus serum has proved of great value, especially during epidemics, but all brands of serum do not seem equally efficacious. In the Edinburgh epidemic (1907), Claude B. Ker³ made use of Flexner's serum with much success. He estimated that if the injections were made within the first three days the mortality was not likely to exceed 16 per cent., and the figures quoted above from Holt (p. 790) are in support of this contention. In sporadic cases serum treatment is not so successful.

In using the serum, lumbar puncture is done, and an amount of cerebro-spinal fluid slightly greater than the serum to be used is withdrawn. The serum is then injected very slowly with the usual precautions, and the foot of the bed is kept raised for some hours. If there should result headache, as

¹ S. M'Lean and J. P. Caffey, *Journ. Amer. Med. Assoc.*, 1926, lxxxvii., 91.

² P. L. Du Bois and J. B. Neal, *Amer. Journ. Dis. Child.*, 1915, ix., 1.

³ Claude B. Ker, *Manual of Fevers*, London, 1911, 300.

does not infrequently occur, morphine may be administered. The dose of serum in the case of children varies from 15 to 30 c.c., and the injections are repeated daily for four or five days. A persistence of fever, marked leucocytosis, and of organisms in the fluid calls for a continuation of the injections. If there is a diminution of the cells and a disappearance of organisms from the fluid, with general improvement in the patient's condition, even although there is slight fever, the injections may be discontinued. It must be remembered, however, that the injections *per se* may cause a rise of temperature.

It has been remarked above that occasionally no organisms are present in the fluid obtained either by lumbar or cistern puncture, and that in these circumstances ventricular puncture is indicated. For the same reason it may be necessary to administer the serum per the ventricular route. This should always be done if organisms are present in the ventricle, as it is only in this way that the serum can reach them. The same procedure is also necessary if the fluid is thick, if little fluid is obtained by lumbar puncture, or if there is no sign of improvement after several doses of the serum have been given by the lumbar route.

M'Kenzie and Martin¹ have pointed out that better results are obtained when anti-serum is given along with some fresh human serum. This they ascribed to the addition of complement normally present in fresh serum and absent from cerebro-spinal fluid, and which is essential if the anti-bodies of the curative serum are to exert their effect. In obstinate cases this method of increasing the potency of the serum therapy should certainly be tried, as the death-rate is still alarmingly high.

Good results have sometimes been obtained from intravenous injection of serum in the early stages, and in chronic cases Claude B. Ker reports that autogenous vaccines are occasionally of benefit.

Acute Suppurative Meningitis.

Acute purulent meningitis is not uncommon, especially in the first year of life. It sometimes follows an injury, and may be due to infection by staphylococci, streptococci, or other

¹ I. M'Kenzie and W. B. M. Martin, *Journ. Path. and Bact.*, 1908, xii., 539.

pyogenic organisms spreading from neighbouring parts, as in the case of suppurating sores, disease of the petrous bone, erysipelas, etc. In the majority of cases, however, it is caused by the pneumococcus. When this is so, the meningitis may be secondary to pneumonia, empyema, or otitis; but this is not always the case. Suppurative meningitis may also be due to the influenza bacillus and in the new-born to the *Bacillus coli*. The distribution of the lesion varies in different cases, but generally the vertex is severely affected.

Symptoms.—The course of the disease is very acute in most cases. It is generally fatal within a few days, or, at most, within a week. Occasionally, however, especially in older children, it may last longer; and in a very few cases the child may recover.¹ This is specially true of influenzal meningitis. Rivers² found the average duration in 220 cases of influenzal meningitis to be 18 days: some cases lived for several months: of cases under two years the mortality rate was 97 per cent., and when over two years of age only 71 per cent.

The onset is sudden, with high fever, vomiting, headache, delirium, and giddiness, and there are generally convulsions. The fontanelle bulges, there is usually head retraction with general rigidity and tenderness, and often strabismus and nystagmoid movements of the eyes. Coma supervenes early, and there is sometimes hyperpyrexia (107° to 108°) before death. There is usually a high degree of leucocytosis.

In cases where the vertex is the part chiefly affected, the symptoms are often very obscure; and without lumbar puncture the diagnosis may be impossible. In such cases there may be no head retraction, and the pyrexia, vomiting, and stupor might accompany any high fever. Purulent meningitis occurs, not very rarely, as a terminal complication in weakly babies, and in them it may cause almost no symptoms.

The diagnosis of the special type of meningitis depends on the bacteriological examination of the cerebro-spinal fluid.

Treatment.—Should any primary source of suppuration be found, it should, of course, be dealt with at once. In strong children when the distress is great, the application of one or two leeches behind the ears gives relief in the early stages

¹ W. F. Fedden and G. M. Kendal, "Case of Otitic Cerebro-spinal Meningitis," *Brit. Med. Journ.*, 1924, ii., 323 (literature).

² T. M. Rivers, *Amer. Journ. Dis. Child.*, 1922, xxiv., 102.

Generally, we apply an ice-bag to the head and give urotropine (5 to 10 gr.) every four hours; also bromide and chloral or inhalations of chloroform when convulsions threaten. Prolonged hot baths (95° to 100° F.) are also useful. Mercurial inunction seems sometimes beneficial. Lumbar puncture may lessen the pain, and, if so, should be frequently repeated. In cases which survive the first week or so, the treatment is the same as that for posterior basic meningitis. Serum treatment is sometimes successful.¹

Serous Meningitis.

In certain cases there is no doubt from lumbar puncture that there exists an inflammatory condition of the meninges, but organisms cannot be detected in films or isolated by culture. Sometimes the cells are chiefly lymphocytic and at other times almost wholly polymorphonuclear in type. In the former case tuberculous meningitis is naturally suspected, but the fact that the child makes an uninterrupted recovery after lumbar puncture removes entirely such a possibility. A sterile fluid containing an excess of cells and globulin is found in cerebral abscess, and also in otitis media, but neither of these conditions can explain the meningeal reaction in the vast majority of the cases. It has been suggested that they are examples of benign or very mild meningococcus meningitis, as this is the only type of meningeal inflammation which is generally recoverable from. Many of these patients are, however, acutely ill, severely toxic, suffer from convulsions, and go into coma, and hence it is difficult to ascribe such grave manifestations to a benign or mild infection. The fact, too, that even very early in the course of the disease the exudate may be almost entirely lymphocytic in type is also against a meningococcal etiology.

Whatever the cause, there is no doubt about the existence of such a condition and the rapidity with which recovery takes place after lumbar puncture. This points the lesson that one should never dogmatise on the nature of meningitis, and hence also the probable outcome, in the absence of a bacteriological finding. And further, it teaches us that just as lumbar puncture is essential for diagnosis it may have a very definite curative value.

¹ C. W. Vining and H. P. Thompson, "Case of Streptococcal Meningitis treated with Serum : Recovery," *Brit. Med. Journ.*, 1924, ii., 667.

Pachymeningitis Hæmorrhagica (Idiopathic).

It is appropriate that mention should be made here of a peculiar affection of the meninges only met with during infancy, and which in many instances closely simulates meningitis.

Its exact pathology is undecided. Although called pachymeningitis, Finkelstein¹ considers that it is not a disease of the dura mater but one of the pia-arachnoid. It is variously ascribed to inflammation and to a new formation. According to the latter view the condition starts as an exceedingly vascular membranous new formation on the inner aspect of the dura, or outer aspect of the pia-arachnoid, and that the hæmorrhage and exudate are accidental; while those who hold the former opinion consider the hæmorrhagic exudate primary and the membranous formation secondary. In any recent case, however, a membrane has always been found on the inner aspect of the dura mater and extending therefrom into the pia-arachnoid as a loose tissue enclosing cystic spaces of varying size filled with a hæmorrhagic fluid. The fact that cerebro-spinal fluid obtained by lumbar puncture may be absolutely clear and devoid of any abnormality, is not in favour of an inflammatory process and points to the mischief being sharply delimited. The cerebro-spinal fluid surrounding the spinal cord may, however, be blood-stained or xanthochromatic.

In situation the lesion is usually bilateral, a favourite site being on each side of the *falx cerebri* over the parietal lobes. The posterior fossa would never seem to be involved.

The *etiology* is obscure. Lues has occasionally been associated. In none of our cases was the Wassermann reaction positive. The condition would not seem to have any relationship to the hæmorrhagic diathesis, and the sterility of the exudate speaks against an infection, although it has been noted to occur after broncho-pneumonia and whooping-cough. In most of the cases under our observation there was no history of any previous illness. There is no evidence that thrombosis of the cerebral sinuses plays any part in its causation, although we know that thrombosis following otitis media, and occurring as a late event in marantic infants, may produce meningeal hæmorrhage.

Clinically two varieties of the condition are met with,

¹ H. Finkelstein, *Lehrbuch der Säuglingskrankheiten*, Berlin, 1905, p. 226.

(a) *the acute or meningitic form* in which there is a sudden onset with screaming, fever, convulsions, nuchal rigidity, unconsciousness and bulging of the anterior fontanelle, and (b) *the chronic variety* in which the child gradually becomes ill without any characteristic manifestations, until bulging of the anterior fontanelle makes its appearance. The clinical picture may simulate a developing hydrocephalus. Finkelstein describes a third variety in which the disease commences insidiously, but in which there occurs later a sudden and acute exacerbation, probably in consequence of hæmorrhage.

In both the acute and chronic varieties, *papillædema*, *optic neuritis* and *retinal hæmorrhages* may be observed. This might at first sight suggest a generalised toxæmia, but the probable reason is the sudden accession in intracranial pressure. In all cases there is a tendency to hypertonus of the limbs with increased reflexes.

The *diagnosis* rests on the result obtained on puncturing the bulging anterior fontanelle. If a needle is inserted laterally, as if to tap the ventricle (p. 787), it will be found that, so soon as the needle has reached a depth of a few millimetres, a markedly blood-stained or xanthochromatic cerebro-spinal fluid escapes. Indeed, the condition may be an accidental finding when attempting to do ventricular puncture. The amount of fluid varies much—at times 25 c.c. or more can be easily withdrawn. Lumbar puncture, on the other hand, may reveal an absolutely normal cerebro-spinal fluid or one slightly blood-stained or xanthochromatic. It will usually be observed that the withdrawal of fluid by the lumbar route has no effect on the intracranial pressure, and that the fontanelle continues to bulge.

One must of course differentiate between this condition (idiopathic pachymeningitis hæmorrhagica) and those cases of meningeal hæmorrhage due to birth injury; the onset in the latter circumstances is during the first few days of life, and the cerebro-spinal fluid obtained by lumbar puncture usually contains blood. Pachymeningitis, on the other hand, usually occurs during later infancy, especially from the fourth to the eighth month. As previously mentioned, thrombosis of the great sinuses may also cause extravasation of blood into the meninges: the seat of the hæmorrhage in these cases, however, bears some relationship to the sinus involved, and thus, when

the cavernous sinus is affected, the hæmorrhage may be situated in the anterior fossa behind the orbits and cause proptosis, whereas in the case of lateral sinus thrombosis the effusion of blood may be in the posterior fossa, which always escapes in the idiopathic pachymeningitis.

The *prognosis* is uncertain. Many of the children die, but others make an apparently complete recovery, although in some cases evidence of cerebral damage persists. This is equally true of both the acute and chronic varieties of the condition.

Treatment is entirely symptomatic, but withdrawal of the fluid has seemed in some of our cases to hasten recovery.

Chronic Hydrocephalus.¹

Symptoms.—The most noticeable thing about a case of chronic hydrocephalus is usually the alteration in the shape and size of the head. This has been already described (Figs. 32 to 37, p. 67). As the head continues to enlarge, the sutures gape and sometimes, especially when the head is not enlarging rapidly, small rounded areas of craniotabes make their appearance in the parietal and occipital bones. When the enlargement is slow there may be severe headache and vomiting, owing to an advanced state of ossification of the cranium, but in the rapidly growing cases in babies these symptoms are usually absent. The increasing displacement of the roof of the orbit causes a downward direction of the eyeballs, so that the sclerotics over the iris become exposed, and in time the pupils lie behind the lower lids.

In a few cases the sight is permanently lost from optic atrophy, but optic neuritis is rarely found. Often there is temporary blindness due to the heightened pressure, which passes off when the fluid ceases to increase. In many cases the sight seems unaffected. Occasionally other cranial nerves are paralysed, and this may give rise to an erroneous diagnosis of cerebral tumour. The limbs often show a degree of ataxia, sometimes there is head retraction (Fig. 215, p. 681), and not uncommonly severe spastic diplegia. In a considerable number of the congenital cases of hydrocephalus, spina bifida is also present.

¹ Professor John Fraser, who originally wrote the sections on the Causation, Pathology, and Treatment of Chronic Hydrocephalus, has kindly revised them for the present edition.

In cases which are advancing, the fluid varies in amount from time to time; and, when it is on the increase, there may be exacerbations of vomiting and screaming attacks. The screaming is distressing to the parents, but it is probably not due to pain. The cerebro-spinal fluid is generally clear and normal in appearance.

The child's body is often puny and always weak, and in many cases there is a tendency to convulsions.

Mental Condition.—Chronic hydrocephalus is a common cause of mental defect, and if the disease is at all severe in degree the mental condition is always below the average. In some slight cases the defect may be scarcely noticeable, and even in the more serious ones its degree is usually less than might have been expected from the appearance of the head. Hydrocephalic imbeciles are usually gentle and amiable in disposition, unless they have been spoiled, but they are not capable of much education. The prognosis as to mental improvement is generally better in cases that date from birth than in those which have begun in later childhood.

Diagnosis.—The only cases in which the diagnosis is apt to be overlooked are those in which the hydrocephalus is beginning to form rapidly, while the head has not yet begun to enlarge, and there have been as yet no definitely cerebral symptoms. In these cases the irritability, screaming, and vomiting are often attributed to digestive disturbance, until the displacement of the eyeballs and the appearance of craniotabes show their real nature.

Prognosis.—In most untreated cases there is a slowly progressive increase of the symptoms; but instances are not very rare in which the fluid spontaneously ceases to gather after a time, and the child recovers with a permanently enlarged head, usually with a damaged intellect, and sometimes with a degree of rigidity of the lower limbs. So far as our experience goes, most of the syphilitic cases improve or recover under early specific treatment.

Causation.—Within recent years, and chiefly through the work of American investigators, our appreciation of the problem of hydrocephalus has materially altered. We have had to modify our previous ideas of the physiology of the cerebro-spinal fluid circulation; and this has meant that classifications formerly adopted have been abandoned, and that the clinical

aspects, particularly in respect of treatment, have had to be considered upon a new basis.

The Physiology of the Cerebro-spinal Circulation.—Dandy's experiments have shown that the choroid plexuses of the various ventricles are the sites at which the bulk of the cerebro-spinal fluid is produced; a very small proportion is formed in the perivascular spaces which extend throughout the cerebral vascular system, and this source, though responsible for only a relatively small amount of fluid, is yet of interest and importance, for it represents an extra-ventricular source the existence of which was previously unsuspected.

After production, the exact method of which is still doubtful, the fluid circulates along the ventricular system and eventually escapes through the foramen of Magendie and the foramina of Luschka into the large subarachnoid spaces or cisterns which lie at the base of the brain. These represent pressure-stabilising and distributing centres, points from which the fluid is uniformly distributed over the cerebral hemispheres and along the surfaces of the spinal cord.

As the fluid passes over the brain, one point is of particular importance—the space between the free edge of the tentorium cerebelli¹ and the crura cerebri. Through this comparatively narrow area the bulk of the fluid has to pass on its way to the absorption fields of the cerebral hemispheres, and adhesions in this situation may easily constitute a very real obstruction to the circulation of the fluid.

The arrangements which exist for the absorption of the fluid are mainly in the subarachnoid tissue of the cerebral hemispheres; arachnoid villi (the Pacchionian bodies of the adult), arachnoid mesothelial cells and the communications of the perineural lymphatics are the forms which the arrangements take.

The Essential Features in the Development of Hydrocephalus.—Theoretically it may be argued that one of three possibilities may explain the development of a hydrocephalus:—

- (a) An abnormal production of fluid.
- (b) An obstruction to the circulation of the fluid either in its intra-ventricular or extra-ventricular course.
- (c) A diminished power of fluid absorption.

¹ Dandy and Blackfan, *Amer. Journ. Dis. Child.*, 1914, viii., 406; and *ibid.*, 1917, xiv., 424.

In practice we have no evidence that the first or the last of these possibilities occurs, and the view generally held is that all examples of hydrocephalus are the result of an obstruction to the circulation of the fluid.

Classification.—With this understanding it is obvious that a classification must be adopted which expresses the importance of the obstructive influences.

A small proportion of cases may be correctly described as “congenital,” in so far as they are the result of congenital anomalies in the cerebral structures. Cases of this nature are often accompanied by spina bifida, and as a group they are rarely amenable to relief measures.

The remaining cases (and they form the great proportion) are examples of an obstructive origin, whether pre-natal or post-natal in point of time, and according as the obstruction is intra-ventricular or extra-ventricular in position, different types of the disease are recognised. An inclusive classification may therefore be arranged as follows:—

Hydrocephalus	{	Congenital	{	Obstructive intra-ventricular.
		Acquired		Obstructive extra-ventricular.

Pathology.—Omitting consideration of the pathology of the congenital variety of hydrocephalus, as the disease is not amenable to treatment, the pathology of the acquired types is related to the various influences which produce obstructive changes, and thus birth-hæmorrhages, meningitis, congenital syphilis, and cerebello-pontine tumours are possible causes.

It is obvious that the “narrow points” in the topography of the cerebro-spinal fluid circulation are the situations in which pressure influences from any of the above-mentioned causes would become most effective, and therefore we find that the aqueduct of Sylvius, the foramina perforating the roof of the fourth ventricle, the space between the free edge of the tentorium cerebelli and the crura cerebri, and the subarachnoid basal cisterns are the situations in which the obstruction is generally located.

The effect of the obstruction, wherever situated, is to produce a backward pressure, the result of which is evidenced in a

progressive distension of the ventricular system with cerebro-spinal fluid. The distribution of the distension will necessarily depend on the type of hydrocephalus; in the extra-ventricular type the complete ventricular system is involved; in the ventricular variety the distension will depend on the situation of the obstruction.

In a long-standing case the cerebral tissue becomes a mere shell, and it is interesting to observe that the disappearance of the cerebral tissue is at the expense of the white matter; even in the most advanced examples of the disease a grey zone of approximately normal thickness results. The cerebral sulci become opened out, and in the frontal region of the brain all trace of these may disappear. The fontanelles, the sutures, and the imperfect ossification of the cranial vault encourage an enlargement of the skull dome while the more unyielding base is not correspondingly affected. It is this disproportion which gives the characteristic clinical appearance of hydrocephalus, which explains the unlikelihood of there being much displacement of the medulla into the foramen magnum, and therefore the relatively long duration of life which characterises these cases.

The Physical Examination of the Case.—In the majority of cases it is sufficiently obvious that a hydrocephalus is present, but further examination is required if the case under review is to be efficiently classified and intelligently treated. Efficient classification necessarily involves an attempt being made to answer three questions:—

1. Does any evidence exist which would indicate the possible origin of the disease?
2. To what variety of hydrocephalus does it belong?
3. At what level does the lesion exist which is responsible for the hydrocephalus?

The physical examination is directed towards supplying an answer to each of these individual queries so far as is possible. The answers having been given, classification is possible, and an intelligent treatment may be planned.

1. *The Origin of the Disease.*—If this question can be answered, the information will be obtained by careful questioning of the parents, a full case-history, and general examination of the child. Attention is specially paid to three possibilities—

the occurrence of a syphilitic infection, the infliction of an injury to the skull, such as may have produced an intracranial hæmorrhage, and the history of a previous meningeal or encephalic infection. A fourth possibility exists, namely, that of the coincident existence of a cerebral tumour.

In certain cases the evidence obtained is sufficiently strong to enable one to estimate with a degree of certainty the origin of the hydrocephalus, as, for example, in the specific types and the post-infective meningeal conditions.

A considerable proportion of cases, however, necessarily remain in which no definite idea can be formed of the etiology, and such a lack of knowledge is not after all serious, because an answer to the question is not essential in deciding on the line of treatment to be adopted. If possible, however, an attempt should be made to classify the case according to its etiology.

2. *The Type of the Hydrocephalus (Intra-ventricular or Extra-ventricular).*—It is obvious how important it is to decide whether the obstruction exists within the ventricular system or whether it is situated in the subarachnoid and extra-ventricular regions. It is apparent that without this knowledge any treatment must be more or less empirical.

Three methods of establishing this knowledge are at our disposal—lumbar puncture, the intra-ventricular injection of an indicator with the investigation of the cerebro-spinal fluid, and the recovery of the ventricular injection substance from the urine.

(a) *Lumbar Puncture.*—Lumbar puncture may give a suggestive result, but not a definite answer to the question. An intra-ventricular hydrocephalus may show low tension in the spinal fluid, while an extra-ventricular hydrocephalus may show an increased tension; but variations exist, and occasionally the *sequelæ* are reversed. Therefore this investigation cannot be accepted as absolute.

(b) *Intra-ventricular Injection and Investigation of the Cerebro-spinal Fluid.*—If a special fluid is introduced into the distended ventricular area, and if this fluid can be recovered from the spinal cerebro-spinal fluid, it is apparent that no obstruction exists within the ventricles; a negative result is confirmatory of the existence of an intra-ventricular obstruction.

The technique usually followed is that 1 c.c. of phenol-

sulphone-phthalein is injected into one lateral ventricle, using a fine long needle and a small "record" syringe. After an interval of thirty minutes a lumbar puncture is done and the spinal fluid is collected in a test-tube containing one or two drops of 25 per cent. sodium hydrate solution—a rose tint in the alkalinised spinal fluid indicates that the ventricular injection has been recovered and that the hydrocephalus is therefore of the extra-ventricular type.

(c) *The Recovery of the Ventricular Injection Substance from the Urine.*—It is said that normally the amount of fluid absorption which occurs in the ventricular system is negligible; it is estimated at less than 1 per cent. in two hours. The bulk of the absorption within the skull occurs in the subarachnoid spaces, from which area as much as 40 to 60 per cent. of an introduced fluid is excreted after two hours' interval. Based on these considerations, the recovery of phenol-sulphone-phthalein from the urine subsequent to its injection into the ventricles becomes a matter of importance, for, if a ventricular hydrocephalus exists, practically none of the indicator will be recoverable within a reasonable time (two hours), while, if an extra-ventricular hydrocephalus is present, the indicator will be recoverable, though not to the same degree as in a normal case.

Investigation by these methods (and the second is the one adopted as a routine) carries the observer a step forward in so far as he is able to classify the case according to whether it is intra- or extra-ventricular in type.

3. *The Level of the Lesion.*—It is obvious that if there is a possibility of demonstrating the exact situation or level of the obstructive lesion, knowledge of a most important kind from the standpoint of treatment is obtained. The method of oxygen injection followed by X-ray examination of the skull affords this information. The technique is so specialised that it is unnecessary to enter into its details; it is sufficient to say that in cases of ventricular obstruction 100 to 150 c.c. of cerebro-spinal fluid are removed from the lateral ventricle of one side, and the fluid is replaced by a corresponding amount of oxygen. A series of X-ray photographs are now taken, the head being manipulated so as to allow the oxygen to be carried along the various channels of the ventricular system up to the point of obstruction. A wonderfully accurate picture of the patent ventricular

channels is thus obtained. In the extra-ventricular case the oxygen injection may be made into the ventricle or into the vertebral canal and the subsequent accumulation of the gas behind the obstruction may be apparent, but in point of fact the demonstration of the situation of an extra-ventricular obstruction by oxygen injection is less satisfactory than the intra-ventricular procedure.

Treatment.—The treatment of hydrocephalus, in whatever way we may regard it, is difficult and depressing; in fact, there are those who say that, where a *lusus naturæ* of this type exists, the proper and ethical procedure is to remain satisfied with the treatment of such symptoms as may arise, and to avoid interference with the primary error. It has always been our practice, however, to operate on such cases as hold out possibilities of relief, and we feel that in a certain proportion of these some degree of benefit has been secured. One thing is obvious—if anything is to be gained by operation the procedure must be carried through before the condition is too well established; there is no advantage in operating on a child whose white matter is already reduced to a shell.

It is convenient to discuss the possibilities of improvement under the respective headings of medical and surgical treatment.

Medical Treatment.—Medical means of controlling the secretion or absorption of the cerebro-spinal fluid have hitherto met with indifferent success. There is some evidence that the administration of thyroid extract by the mouth diminishes the secretion,¹ but the results of this therapy have been disappointing.

Marriott,² in the course of some observations on œdema, found that certain of the purin diuretics, and particularly theobromo-sodio-salicylate (diuretin) when given by the mouth led to a distinct raising of the blood surface tension. On this basis he argued that the raising of the surface tension of the blood would result in the passage of fluid from the subarachnoid spaces into the blood, and so induce an increased absorption of spinal fluid. Acting on this hypothesis he advises the administration of diuretin to cases of hydrocephalus of the extra-ventricular (communicating) type, and he claims to have obtained satis-

¹ C. H. Frazier, *Amer. Journ. Dis. Child.*, Feb. 1916, xi., 95.

² W. McK. Marriott, *Amer. Journ. Dis. Child.*, xxviii., No. 4, 479.

factory results. The dosage is large—for an infant of six months, 0.2 gramme are administered by mouth three times daily.

Surgical Treatment.—A great variety of surgical procedures have been and still are practised in attempting to relieve this distressing condition. The following Table is a summary of these :—

1. Intermittent drainage.	{ Of lateral ventricle. Of the spinal canal.
2. Continuous drainage.	{ By puncture of the corpus callosum.
A. Of the lateral ventricle.	{ To the surface. To subcutaneous tissue. Into the subdural or subarachnoid spaces. Into the peritoneal cavity. Into the temporal vein. Into the superior longitudinal sinus.
B. Of the spinal canal.	{ To the surface. Into the retroperitoneal tissues. Into the peritoneal cavity.
C. Of the subarachnoid space (fourth ventricle).	
D. Of the cisterna magna into the cranial sinuses.	
3. Indirect treatment (carotid ligatures).	
4. Other methods of treatment, such as by a seton, injection of iodine, galvano-puncture, compression of head, and by drugs.	

Our own experience of the operative measures may be expressed thus. If the hydrocephalus is an intra-ventricular one and the site of the obstruction is reasonably accessible (as for example the roof of the fourth ventricle), the site of the obstruction is exposed and the obstruction removed; if, on the other hand, the situation is inaccessible, we employ the “brush” method, by which a brush drain of silkworm gut passes from the lateral ventricle through the cortical tissue into the subarachnoid space so as to establish permanent drainage.

If the hydrocephalus is extra-ventricular in type we employ the method of ligature of the common carotids,¹ an interval of two weeks elapsing between the two operations. This procedure appears to definitely diminish the production of cerebro-spinal

¹ H. J. Stiles, *Trans. Med. Chir. Soc. Edin.*, 1905, xxiv., 187.

fluid, and so brings the ratio of production and diminished absorption into greater uniformity.

In view of the fact that in extra-ventricular hydrocephalus the obstruction is frequently situated between the tentorium cerebelli and the crura cerebri, the operation of neostomy—the opening of a fresh channel through the tentorium lateral to the obstruction—has been suggested, but we have had no practical experience of the value or otherwise of this method.

CHAPTER XXXII

MENTAL DEFECT (*AMENTIA*)

LIKE "paralysis," "fever," and "diarrhœa," the term "mental defect" does not, properly speaking, designate a disease, but only a symptom which may be caused by many diseases. It is, however, from its nature, such an overwhelmingly important symptom that it overshadows all the other manifestations of disease that are present, and the children who show it are apt to be regarded simply as idiots or imbeciles, irrespective of the difference in the lesions which have made them so. The words "idiocy" and "imbecility" have, however, such unpleasant associations, that they should never be used in the hearing of the child's relatives. "Amentia" is a useful term, which includes all degrees of imperfect or arrested mental development.

Classification.—In attempting to classify cases of mental defect we have two quite different ends in view. These are (1) the *certification and grading* of the children for special classes and for training and custodiary institutions; and (2) an *increase of our knowledge* of the various diseases which cause amentia, and of the clinical features associated with each of them. We cannot at present frame any single classification which will answer both these ends. The first requires mainly a grouping according to the *degree* or *character* of the mental defect, while the other calls for a classification that takes into account all *the pathological and clinical abnormalities* in the cases. We have, therefore, to use two forms of classification.

The *first* of these is represented by that laid down in the Mental Deficiency Act (1913). This aims simply at arranging the children in groups according to the degree of mental defect present (as a librarian sorts his books by their sizes), and ignores altogether the nature of the abnormality which is making them different from other children.

The Act divides all cases of mental defect into four classes

and supplies authoritative definitions of the meaning we are expected to attach to each of the terms which are in general use. These definitions are as follows:—

(a) Idiots ; that is to say, persons so deeply defective in mind from birth or from an early age as to be unable to guard themselves against common physical dangers.

(b) Imbeciles ; that is to say, persons in whose case there exists from birth or from an early age mental defectiveness not amounting to idiocy, yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so.

(c) Feeble-minded persons ; that is to say, persons in whose case there exists from birth or from an early age mental defectiveness not amounting to imbecility, yet so pronounced that they require care, supervision, and control for their own protection or for the protection of others, or, in the case of children, who are incapable of receiving proper benefit from the instruction in ordinary schools.

(d) Moral imbeciles ; that is say, persons who from an early age display some permanent mental defect coupled with strong, vicious, or criminal propensities on which punishment has little or no deterrent effect.

In America the term “feeble-mindedness” is used in a wider sense than in this country, and denotes any degree of mental defect ; and the word “moron” is applied to the milder cases which we call “feeble-minded.”

The *second* form of classification is illustrated in the accompanying Table taken from Dr Tredgold’s book.¹ It is, in

Etiology.		Clinical Varieties.
PRIMARY AMENTIA.	A numerical deficiency, irregular arrangement, and imperfect development of cortical neurons.	1. SIMPLE AMENTIA. 2. MICROCEPHALIC. 3. MONGOLISM.
SECONDARY AMENTIA.	I. { GROSS CEREBRAL LESIONS.	4. CONGENITAL SYPHILITIC. 5. AMAUROTIC. 6. HYDROCEPHALIC. 7. PORENCEPHALIC. 8. SCLEROTIC. 9. PARALYTIC.
	II. { DEFECTIVE CEREBRAL NUTRITION.	10. Other TOXIC, INFLAMMATORY, and VASCULAR. 11. EPILEPSY. 12. CRETINISM. 13. NUTRITIONAL. 14. ISOLATION.

¹ *Mental Deficiency*, London, 3rd edit., 1920, 99.

some respects, a modification of that drawn up by the late Dr Ireland, and is the most useful that we know.

In this Table there is an important division of the cases into two great groups: (*A*) The *primary* cases, which depend on an original incapacity of the cerebral neurons for proper development; and (*B*) those in which the defect is *secondary*, being due to the arrest of the cerebral development at a later stage by some extraneous or accidental cause. In the latter, the cause to which the defect of the neurons is secondary may have occurred during intra-uterine life, at the time of birth, or during early childhood.

The primary group is said to include from 85 to 90, and the secondary 10 to 15 per cent. of all the cases. This refers to children of school age. Among cases seen in infancy, the proportions of the two groups would probably be better given as 60 and 40 per cent. respectively. The difference is due, partly to the large number of the secondary cases that die during the first few years of life, and partly to the great difficulty there is in recognising slight cases of the primary variety during early childhood. Within recent years the epidemics of encephalitis lethargica have caused a great increase in the numbers falling into the secondary group (p. 770).

Most of the forms of disease which seriously damage a child's mental powers also lessen his resistance to disease; hence a large proportion of mentally deficient children die within a few years of birth. We see, therefore, many more infants than older children affected in this way, and many more children than adults.

Before considering the more important of the different types of these children, we shall deal with the symptoms and treatment of the condition in general.

Symptoms of Amentia.

The *recognition of mental defect* in a baby depends partly on what his mother tells us about him, and partly on what we notice for ourselves.

(1) From the *mother's account* we may learn many facts which bear more or less on the child's condition. She may, for example, give a history of insanity or alcoholism in the family, or of mental defect, extreme nervousness, or convulsions

in other of her children; or she may have had a number of miscarriages, or ill-health during her pregnancy. In many cases the labour has been premature, or abnormal in some way. The mother may also give us the important information that the baby showed signs at birth of having been severely injured during his passage into the world, or has suffered at the time, or later, from convulsions or other significant nervous symptoms (p. 727).

For the necessary account of the *baby's progress* since birth we have also always, of course, to depend on the mother's memory and intelligence.

If the child is even slightly mentally defective, we can usually find out from her that his *behaviour* has in many ways been different from that of other infants. He has probably not shown all the usual actions and movements of normal babyhood, or has been far too long in doing so. Thus, in a severe case, the baby may have been very apathetic—showing little or no desire to take the breast or bottle, and not crowing and making springing movements with his limbs, as most babies do when their mothers sway them up and down in their arms.

In many of the extreme cases the baby has shown little of the normal contentment of the healthy infant, and has spent most of his waking hours in miserable, disconsolate crying without apparent cause. On the other hand, he may have been unduly quiet, so that the mother may remark that "one would not know there was a baby in the house."

Often also he has paid no attention to the sights and sounds around him, and been very long in responding to his mother's touch, voice, and smile; and he may not have seemed to know her from a stranger.

In less severe cases the child may have shown few or none of these gross abnormalities; but it has been noticed that, though neither rickety nor paralysed, he has been far too long in learning to raise and balance his head, to sit up, to stand, to creep, and to walk; and that he has taken no interest in his own limbs or in touching and grasping things. In the case of an older child we may be told that he cannot take a biscuit or a spoon in his hand to feed himself, or drink from a cup without assistance.

Generally the child has more or less complete incontinence of urine and fæces, and does not let his mother know when he needs help in these matters. In some cases the tongue has

been kept constantly protruded, and in many the saliva has dribbled continually from the mouth. Speech has usually been long in coming, if it has come at all.

Another class of symptoms of which the mother may tell us, and which may bring us to an immediate diagnosis, is the occurrence of a *series of epileptiform attacks*. If, for example, there is a history of frequently recurring convulsions continuing for a long period without apparent cause, the presence of mental defect may be regarded as more than probable.

Many of the convulsions which occur in early childhood have, of course, little or no significance with regard to the patient's mental condition, and usually, even when severe, indicate only some temporary disturbance of his bodily health. When, however, the fits are severe as well as frequent, and occur without ascertainable cause, we may be sure that a serious condition of the brain exists which is either causing the seizures or resulting from them.

The persistent recurrence of slight *petit mal* attacks in a baby is a very ominous sign, especially if, as often happens, it is accompanied by an arrest in the normal growth of the cranium. In these cases, although the attacks are commonly so slight at first as not to alarm the mother, the prognosis is extremely grave. No treatment has practically any effect on them, and the resulting mental deterioration is profound and permanent.

(2) *When we examine the baby* we may find, especially in severe cases, that either his *appearance or his behaviour* is so peculiar that it at once betrays the defective state of his brain. His head, for example, may be so abnormally small, or his fontanelle so early closed, that his brain cannot possibly be normal in development. The size of the head should never be overlooked in examining any baby for the first time.

In other cases we see at a glance that the patient is the subject of one or other of the *easily recognised diseases* of which mental defect is a constant or almost constant accompaniment. He may evidently be a mongol or a microcephalic, or suffering from chronic hydrocephalus or from a severe degree of congenital spastic diplegia or of cretinism. When this is so, we know that his mental condition is almost certain to be defective, although he may be so young that it is difficult or impossible, as yet, to find fault with his behaviour.

In some instances in which the child's intelligence is of a low grade, although we can find nothing wrong with his bodily conformation, there may be such marked *abnormalities of behaviour* that the presence of mental defect is quite obvious. This may be shown by his unnatural and uncouth gestures and grimaces—rolling his eyes and his head about constantly, throwing his head back with his mouth wide open, paying no attention to anything, or taking fits of meaningless crying or laughter. In others, the baby is quite apathetic, and never laughs or smiles at all.

When such obvious faults in form or conduct are present it is easy to see that the child is mentally deficient. In most of the infants, however, whose brains are abnormal from birth—including nearly all the slighter cases—there is no such shortcut to a diagnosis. In these we must depend mainly on what we are told by the mother about the child's past conduct, and on how far the development of his senses and the growth of his intelligence have kept pace with his age.

Backwardness in the acquisition of these natural actions may of course be due merely to temporary debility accompanying or following bodily illness, and this frequently happens. If, however, they are too long in coming, and the child shows no sign of debility to account for this, we shall probably be right in suspecting the presence of mental defect.

Acquired Mental Dullness.—Delay in mental development, without mental defect, is occasionally met with, and is apt to be mistaken for imbecility. We find it, for example, in weakly cripples as the result of isolation, with lack of teaching or other mental stimulus. Defects of sight and hearing may lead to a similar result. In such cases, when the abnormal conditions are removed, the mind may develop normally. Sometimes the backwardness is due to such debilitating maladies as chronic diarrhœa, severe rickets, or congenital syphilis. These children may look very like imbeciles, but we are aided in our diagnosis if we investigate their condition as to common sense, a feeling of responsibility, and general correctness of behaviour.

Another small group of older children who may be mistaken for mental defectives are those who have temporarily lost their brightness, and sometimes also many of the signs of normal intelligence, as the result of a nervous breakdown. Such a

breakdown may be caused in neurotic children by prolonged worry and overwork, along with a lack of sympathy and affection on the part of those who are looking after them (p. 698).

The *treatment* of cases of recently acquired dullness from ill-health or overstrain is very different from that of mental defect or mere stupidity, for in it the patient is always to be treated as a sick child and not as a pupil. What his brain needs is rest and recreation—not instruction, however skilful. His bodily health must be fostered by a prolonged period of tranquil routine, which should include plenty of sleep, simple amusements, warmth, massage, tonics, nourishing food, and abundance of open air. While the bodily vigour is being restored and the mind left fallow, the child's feelings must also be soothed by kindly mothering and encouragement. The result of such treatment is generally satisfactory ; but in planning the patient's future career, the weakness of his nervous system must be remembered and an occupation chosen which will not throw too much strain upon it.

Treatment of Amentia during Early Childhood.

With regard to the treatment of mentally deficient children, two general statements may be made—

1. The condition is, strictly speaking, incurable in the sense that children, who are mentally defective to begin with, will remain so to the end, whatever is done.

2. There are very few mentally deficient children who are not capable of being improved and made far happier by suitable treatment.

These two statements are equally true, but the latter is much the more important as a basis of action.

The presence of a mentally defective child in a family is always, sooner or later, the cause of much distress. It is the duty of the medical man to do what he can to lessen this ; and in his treatment he must consider not only the child who is causing the distress, but also the parents who are feeling it.

When a baby is mentally defective, and his parents do not know it, the first question that arises is : Are we to tell them ? If they ask plainly, they must, of course, be told the truth. If, however, as often happens, they ask no direct questions (either

from ignorance or because they are afraid of the answer), it is usually far better to tell them as little as possible. There are two reasons for this apparently disingenuous course of action—(1) A very large proportion of imbecile babies die early. When this happens, it is clearly well for all concerned when the child's defect has remained a secret known only to the doctor. (2) The parents usually know nothing about the subject of mental deficiency. When this is so, a sudden intimation that it is present in their child, if made before their own observation has prepared them to receive it, is apt to produce unsatisfactory results. They either are annoyed and refuse to accept it, or, if they do so, it causes such discouragement as to paralyse their efforts for his improvement.

In most cases, all that it is necessary to tell the mother about the *present* is that her child is unlike the ordinary run of babies in not being able, as yet, to do certain common things as well as they do; and—as to the *future*—that, with patience and careful training, he will come to do many of these things; but that only time will show how far his improvement is likely to go—also, especially, that his progress will largely depend on how much trouble she takes in training him. It is by trying to make the child do things better that she will come most naturally to realise the true state of matters.

The object of our treatment, so far as the child is concerned, is to make him *as happy and as good as possible*. As his happiness will largely depend on how many things he can and does do and notice, and on how far he commends himself to others by his behaviour, our chief aim is to make him more capable and more likeable. His mother must also try to give him a sense of duty proportionate to his intellect, and to show him that his duty lies in doing things that he is quite able to do.

The extent to which mentally defective children benefit from treatment, and the best measures to employ, vary greatly according to the degree of the defect and the nature of the case. In many instances the improvement under bodily and mental culture is remarkable; and, even in the types of idiocy lowest in habits and capacity, the child may benefit very much from an intelligent mother's early training.

Such treatment and training as the child needs during the first few years must, of course, be given by his mother. No one

else can give it him. All that the medical man can do for her is to start her on right lines, and then find a child-welfare visitor or other competent person who will take in hand to visit her regularly, guide her in the training of the child, and encourage her in the carrying out of its details.

If the mother is at all intelligent it is usually a good thing to give her a few printed suggestions. Those used in the Edinburgh Children's Hospital will be found in Appendix D.¹ When she has mastered these simple rules, and is trying to carry them out, it is well to let her have also a simple manual giving fuller details.² From a small book of this kind an educated mother may learn most of what she needs to know. But in the case of many working-class women, and some others, too, who find it perplexing to learn practical details from a printed statement, it is quite necessary that an understanding visitor should explain the directions, week by week at first, until their practice has become a matter of every-day routine. All such directions must be carefully worded so as to leave the child's mental condition an entirely open question, otherwise they may not be of use to those mothers who do not see that their child's mind is affected, and to those who suspect and resent such an idea.

Details of Treatment.

I. The first thing that we have to do is to encourage his mother to keep on trying *to make the baby please her* by practising, again and again, any of the ordinary little actions of infancy which his natural instinct is too long in teaching him. As his muscular power develops, she must find out what actions he has least difficulty with and *likes doing*, and encourage him to repeat them over and over again. Doing these things better will give him confidence, and lead him on to try other things also. He should be specially encouraged in anything that is a little difficult for him. Any success he has

¹ This leaflet may be obtained from the Central Association for Mental Welfare, 24 Buckingham Palace Road, London, S.W. ; price 4d. a dozen.

² A small simple manual called *Opening Doors*, for the mothers of babies who are long in learning to behave like other children, may be obtained from the Publishers, Messrs Oliver & Boyd Ltd., Tweeddale Court, Edinburgh, and 33 Paternoster Row, London, E.C. ; price 6d., and 5s. per dozen.

in such things will give him special pleasure, while trying vainly to do what is quite beyond him can only discourage him.

The difficulty mentally defective babies have in using their limbs for any purpose may be of two kinds.

In most instances the child does too little *because he does not try*. His muscles are normal, though weak from want of use; and he does little with them, because he has not yet felt the normal infant's keen desire for movement. When this is so, the mother's aim must be to arouse his interest in outside things in every possible way.

In other children—those, namely, with *spastic paralysis*—the muscles have no lack of strength, but their movements are hampered exceedingly by the general rigidity of the limbs and the consequent difficulty the child has in getting them to obey his will. In a fair number of these babies the will-power is quite strong, so that there is a good prospect of improvement from persevering training. In many, however, the mental condition is of such a low grade that their weak wills are powerless against the stiffness of their limbs, and they prove hopelessly ineducable.

When the children are capable of being amused by such simple toys as a rattle, a soft ball, or an india-rubber balloon, these things may be useful in attracting their attention, and tempting them to move their limbs. Any toys that can be blown through, such as a whistle or a trumpet, have the further advantage that they tend to strengthen the lips of those children who dribble too much.

The baby should be specially encouraged and helped to do anything harmless that he does of his own accord; for such things please him far more than any little games suggested to him by others. If he wants to do something, tries hard to do it, and at length succeeds, this is a most valuable as well as a very pleasant lesson for him. As he grows older, the mother must be careful never to go on doing for him anything, however small, that he can possibly do for himself. If he likes to hear his rattle, he should be persuaded to shake it; and if the movement of his balloon pleases him, he should hit it himself to make it move.

II. The mother must be instructed always to watch for and check at once any *bad habits* the baby begins to practise. Even very dull babies can often be made quickly to understand when

their mothers do not like what they are doing and mean them to stop it. These unpleasant actions, once they have been allowed to grow into regular habits, are far harder to stop in these infants than in normal children who have plenty of outside interests. It is also important that, when a baby is checked for doing anything of this sort, his attention should always if possible be attracted at the same time to something else. Such habits gain a hold on a child chiefly when his mind is turned in on itself, because he is not being interested in outside things.

III. If the baby is *taking too little notice*, he must be encouraged to look at, listen to, and handle anything that he is taken up with. If watching the people, horses, and cars that pass the window does not interest him as yet, perhaps bright lights, brilliantly coloured pictures and toys, or music may. Anything that interests and pleases him will tend to make him brighter and more noticing. When his mother finds something that he likes, she should let him have it often, and encourage him to keep his attention fixed on it as long as possible. When the baby is long in showing a special interest in his mother, she should take pains to draw his attention to herself by talking to and playing with him, and trying to get him to look at and smile to her.

IV. As we have seen, the mentally defective child is nearly always very *slow in learning to speak*. This is partly due, of course, to his being so long in paying any attention to what his mother says to him that he does not learn the meaning of her words. He should be spoken to constantly from the first, even though he seems to pay no attention to what is said. The normal baby only learns the meaning of words by having them repeated to him an innumerable number of times. Even after they understand everything that is said to them, these children often require a great deal of encouragement before they begin to say anything themselves. Once they begin, however, to use a few words or even inarticulate sounds with a definite meaning, their speech will continue to improve; but it will very often remain indistinct and slurred in character.

The mother should begin by getting the baby to use his voice in imitating simple musical or other sounds, and then to say short easy words after her. She should repeat these to

him over and over again through the day, and especially when he awakes in the morning and before he goes to sleep at night. When she names things she should show them to him, and try to make him understand the meaning of other words, such as "up" or "down," by her actions. A great deal of patience is often needed in this matter. Some children, who will not learn to speak at all during early childhood, do so rapidly and well when they are older.

V. As the baby's intelligence grows, his mother must do her best to *develop his character* by increasing his self-control and making him depend on himself as much as possible. The more he does for himself the happier he will be, and the less chance there is of his becoming self-centred and miserable. As he grows older he must be made to understand that his being not quite like the other children does not excuse him from doing, if he can, the little irksome things which they have to do.

Patience, good temper, and good manners are other matters in which the baby's command over himself can be cultivated; but he must not be too much tried by others teasing him.

Prompt obedience is always to be aimed at, but his mother must try to get him to obey more because he wishes to please her than from fear of what might happen if he were disobedient.

It is very good for the baby's character, and for his happiness, if he can be made fond of those about him. Few things brighten a child's life as much as having a warm affection for other people—or even for animals and dolls.

Another matter which has no small importance in connection with the formation of the baby's character is *the control of his bladder and bowel*. The age at which he acquires this depends largely on the training his mother has given him. The emptying of these organs in all babies takes place at regular intervals. The mother must try to find out the length of these intervals in her child's case, and lift him regularly at the right times so as to form a good habit. If such training is carried out with sufficient patience and perseverance, nearly all mentally defective children can be taught cleanly habits, and the effect this has on their character and happiness is very great.

Another matter in which education is often needed is the difficulty some of the children have in taking their meals.

They often refuse to swallow solid food when it is first given them, and it may take a great deal of trouble to induce them to do so. If no trouble is taken about this, they may have to be kept on liquids only, to an extent that is not good for their health. Many of them also can only be taught with great difficulty to chew their food. If they do not learn to do so, it leads to dental caries and sometimes to troublesome dyspepsia.

VI. The habit of *constant, apparently causeless crying* cannot be controlled by medicine, but being much in the open air often lessens it greatly.

Most forms of *epileptiform convulsions* are also very intractable in these children. Occasionally, however, bromide, borax, or luminal have a temporarily good effect.

VII. In cases of *spastic diplegia* it is very important that, in addition to the treatment already discussed, special mechanical measures and exercises should be used to help the children to gain more control over their stiff limbs. This is always a tiresome business for the mother, as it has to be persevered in so long and occupies so much time; but it is generally well worth the trouble it takes. It does what no other kind of treatment can do, and makes a great difference in the long run in the child's capacity for enjoying life.

In beginning this treatment, one difficulty which is sometimes met with is that the infant resents the exercises and movements and cries a great deal. This is probably not due to pain, but only to his strong objection to being bothered to exert himself when he does not wish to do so, and as he gets used to the process the difficulty often lessens.

The special treatment consists in two things:—

(1) The first of these is *detailed passive movement of all the stiff joints*, one by one, to stretch the contracting ligaments and tendons, so as to make them more easily moved, and to prevent or minimise the tendency to contracture and deformity. These movements should be carried out gently but firmly several times a day for some minutes, at a time when the child is feeling restful and cheerful. After a warm bath is a good time for them.

(2) The other part of the treatment consists in encouraging the baby to *overcome the stiffness of his muscles by the force of his will* and to form helpful habits of movement. These exercises must be repeated regularly, at least twice a day.

They also must be carried out quietly, without any fuss, at a time when the infant is feeling happy and at ease. They should never be attempted when he is excited, for any excitement at once tightens all the muscles and makes their movements more difficult to control.

As the child begins to gain more command over his limbs, simple games should be devised to induce him to move them more freely. When he begins to show any desire to make walking movements, for example, the use of a go-cart, and as he grows older of a tricycle, may help him considerably in gaining more efficient control over them. It is always better for the child to make use of such mechanical aids for himself than to be held up by his mother when he is trying to use his limbs.

Ordinary *massage* of the stiff limbs is of no use. It is increased suppleness, not greater strength of the muscles, that is wanted. *Electrical treatment* for the same reason is to be avoided. *Surgical operations* on the muscles and tendons are often helpful, but only when the child is old enough, and sensible enough, to have gained a fair amount of voluntary control over his movements.

VIII. It is scarcely necessary to say that our interest in the infant's mental progress must not be allowed to make us overlook *the state of his bodily health*. Careful regulation of the diet, bathing, and plenty of open air, are most important; and specially warm clothing is generally essential, as the child will always be duller when he is cold.

Such then are the chief details of the home treatment to be recommended in these early cases. There can be no doubt whatever that, when carried out conscientiously, they greatly increase the children's health, happiness, and capacity, and make their later training in institutions, or elsewhere, far easier and more successful than it would otherwise have been.

The children's later training has to be continued on similar lines, and need not be considered here. Full details will be found in the standard works on the subject.¹ An excellent little

¹ Such as *The Mental Affections of Children*, by W. W. Ireland, London, 1898; *Mentally Defective Children*, by Shuttleworth and Potts, 5th edit., London, 1922; *Mental Deficiency*, by A. F. Tredgold, 3rd edit., 1920; *Feeble-Mindedness in Children of School Age*, by C. P. Lapage, 2nd edit., Manchester, 1920.

book on the training of young children has been published by Miss Margaret MacDowall,¹ which will be found most helpful by intelligent mothers.

The Question of Institution Treatment.—To most mentally defective children under six or seven years, in satisfactory homes, a mother's care and affection mean more than anything that the best institution can supply; and after this age there are some cases happily situated of whom the same is true, and who also, like the younger ones, are better off at home than anywhere else.

In the great majority of instances, however, there is much to be said in favour of the child being sent early to a suitable institution or special boarding-school. Many of these children suffer permanently from this not being done.

In discussing what should be done with the child, there are various considerations which have to be taken into account.

1. *The Home Conditions.*—The interests of the parents and those of the other children have to be considered as well as those of the child. Although his presence in the house may do no direct harm to his brothers and sisters, he often makes such demands on his mother's time and energy that she cannot give enough attention to the other children. As the mentally defective child grows older, also, the association with his normal playmates may become a constant source of discouragement to him, because he finds himself hopelessly inferior to them at every turn. Further, unless the house is so situated as to afford a considerable amount of privacy, and the mother has extra assistance, it is often impossible for him while staying at home to enjoy the freedom and open-air exercise that he needs for his health and happiness.

If the home arrangements are not altogether satisfactory, the strain on the mother is apt to be very great. She is probably suffering severely already from the shock of her growing realisation of the child's infirmity; and her strained nerves tell inevitably on him, making him more restless and difficult than he would be in charge of someone who felt less keenly about him.

¹ *Simple Beginnings in the Training of Mentally Defective Children*, London, Local Government Press Co. (R. T. Peach), 1919. The reader will also find wise and helpful suggestions in the Appendix by Miss Mary Dendy in Dr C. P. Lapage's book.

2. *The Child's Present Needs.* — All mentally defective children need protection from the difficulties, dangers, and distresses of life. During the early years a well-ordered home supplies all that is needed in this direction; but often, as the child grows up, it becomes increasingly difficult if not impossible to shield him, or her, from trouble while living at home.

For higher grade mentally defective children, skilled training—mental, physical, and manual—is very important. It helps in developing their intelligence and increasing their feeble initiative, and brightens their lives by making them more like others. Such training can, in most cases, be much better provided in a special boarding-school than at home. Life in an institution has also the great advantage that it allows the child to associate on equal terms with companions like himself, and to have the great and wholesome pleasure of excelling some of his playfellows in something or other. The anxiety which some parents feel lest their child should be deteriorated by living with others less intelligent than himself is quite groundless, if there is proper supervision.

Daily attendance, for a few hours, at a special school is sometimes proposed as an alternative to sending the child away from home; but the influence of such schooling is very small compared with that of a residential institution.

3. *The Child's Future Needs.* — Even when the home conditions are ideal during early childhood, it may be far the best and kindest thing to send the child early to an institution, unless it is certain that as he grows older he will always be equally well looked after at home or with friends. While he is young, he will generally soon learn to live happily under new rules and restrictions; but this may be very difficult for him if he leaves home for the first time when he is older and his habits have become more formed.

Simple Primary Amentia.

The group of simple primary amentia includes the large majority of mentally defective children. Its size is mainly due to the cases which properly belong to it being far commoner than those of any other type. It is also due, though to a much less degree, to a number of cases, which are not really of the same kind, having been put into it provisionally because their

real nature is, as yet, so obscure that we do not know where else to put them. Various groups of mentally defective children that used to be classed along with simple primary amentia have now been differentiated; and there are still many others which, when further investigated, will require to be placed in separate classes.

The **causation** of simple primary amentia is obscure.¹ There is no doubt that, on the average, intelligence is inherited, but mental deficiency is most certainly not a simple matter of heredity. The rarity of a positive family history, the greater rarity of two examples in one family (we personally have only seen one instance of this), its occurrence in some of the most intellectual families, and the fact that the progeny of mentally defective individuals is, as a rule, normal mentally are ample proof of this statement. In illustration of the last point Clarkson² draws attention to an analysis by Fernald of Waverley, Mass., U.S.A., of 1537 cases that had been discharged against his advice from his institution during the course of twenty-five years. 279 had disappeared and 612 had been transferred to other institutions. Of the remaining 646 there were 470 men and 176 women. 27 women had married and had 50 children—33 of these children were living and normal; 7 of the women had no children. There were 11 unmarried mothers, 8 being morons and 3 imbeciles; all the children were normal. Only 13 of the men had married and they had 12 children; 6 had no children; none of the children was defective.

The birth of a mentally defective child, apart from injury and other definite cause, would seem to be more of a sport of Nature. The development of the ovum is such a complex process and is subjected to such a multitude of factors, which act and interact, that the marvel is that mental deficiency is so rare and not much more common. The forms of life on which many of the eugenic experiments have been carried out are so simple, and the factors so controllable, that analogies with the complex question in the human subject are not permissible. The whole subject has been aptly summed up by Jennings in the following sentences: "So long as bi-parental inheritance is kept up, the variety, the surprises, the perplexities, the

¹ See interesting Report of Mental Deficiency Committee of British Med. Assoc., *Brit. Med. Journ.*, Appendix vi., 25th June 1932.

² R. D. Clarkson, *Edin. Med. Journ.*, 1927, N.S., xxxiv., 74.

melodrama that now present themselves among the fruits of the human vine will continue. Capitalists will continue to produce artists, socialists and labourers; labouring men will give birth to capitalists, to philosophers, to men of science; fools will produce wise men, and wise men will produce fools; who mounts will fall, who falls will mount, and all the mass of problems presented to society by the turns of the invisible wheel will remain." ¹

Symptoms.—As the condition is not, in its lesser degrees, characterised by paralysis or by any gross visible fault of bodily conformation, it is often impossible to recognise it in early infancy. Severe cases, however, are easy to distinguish soon after birth. The babies show too little of the normal automatic movements of healthy infancy, and their other motor faculties and speech fail to develop at the proper times. Often also they obviously do not feel, hear, or see like healthy children. Many of the bad cases do not recognise their own mother's smile; many do not know her voice and only recognise her, if at all, by the way she handles them. Their gestures and behaviour, even as infants, are strikingly abnormal, and if they gain control over their bladder and bowel at all, they are very late in doing so. A large proportion of them have frequent epileptic attacks.

As they grow older, their defects become increasingly obvious. When they learn to talk, their speech is usually more or less slurred and indistinct. In many cases there is a constant dribbling of saliva from the mouth. When they walk, their gait remains clumsy and like that of a younger child. They go with their heads forward and their arms hanging in front; their feet are planted far apart, their pelvis often moves too much in walking and the joints of their lower limbs too little. Their hands are generally flabby and nerveless. They are difficult to teach, because they are unable to concentrate their attention on one subject for any time; and they cannot persevere, even in things which interest them. Many seem to have no common sense at all, and do not realise their position or their proper relation to others. Some, chiefly those who take fits, lack the normal shyness of childhood, and others are morbidly shy and self-conscious. They are apt also

¹ H. S. Jennings, *Prometheus on Biology and the Advancement of Man*, London, 1925, 93.

to show a want of self-control, to laugh and cry without sufficient reason, and to get into sudden unreasonable fits of passion. Many of them, as they grow older, show a persistent tendency to wander from home without any apparent aim.

Diagnosis.—Children of this class, who are but slightly affected, make up the majority of the “Mentally Defective Children” in the Special Classes of the Elementary Schools. Such slight cases are often not recognised as abnormal in early infancy, because they have wits enough to perform passably all the duties required of ordinary babyhood. As they grow up, however, more is expected from them, and their shortcomings become increasingly obvious. They are generally slow in learning to walk and to speak. In outward appearance there may be little amiss, but often their heads are distinctly too small and the frontal region especially poorly developed. Their gait also is apt to show, to some extent, the peculiarities already alluded to.

In the slighter cases the deficiency is mainly noticed, while they are children, from the incompetent way in which they do their school work, both mental and manual. They remain far behind normal children of their own age. If a child who has had good health, and with whose education special pains have been taken, cannot pass the Fourth Standard by the time he is fourteen, he may in most cases, as Dr Clarkson has suggested, be fairly classed as mentally defective.¹

The best method of recognising mental defect is by the application of the Binet-Simon *Intelligence Tests*,² or some modification of them.³ They have been standardised for the English child by Burt.⁴ These tests, however, are only applicable in children over three years of age.

¹ A normal child, under ordinary circumstances, should be ready to be promoted from the Infant Department when he is about seven years old. The average age at each Standard may be given as follows:—

Standard I.	7-8 years.	Standard V.	11-12 years.
„ II.	8-9 „	„ VI.	12-13 „
„ III.	9-10 „	„ VII.	13-14 „
„ IV.	10-11 „		

² A. Binet and Th. Simon, *Mentally Defective Children* (translation by W. B. Drummond), London, 1914.

³ L. M. Terman, *The Intelligence of School Children*, London, 1921.

⁴ C. Burt, *Mental and Scholastic Tests*, London, 1922.

On the average, children acquire information and develop their powers of observation and reasoning at a certain definite rate, and these tests are devised to ascertain at what stage any individual has reached. They give a numerical index to the child's mental capacity, and hence are comparable with the foot-rule and scales in estimating the child's physical development. No one would depend on general impressions for gauging the height and weight of a child, and in the case of mental development general impressions are even more untrustworthy. Severe degrees of mental impairment may of course be appreciated without the aid of these tests, and indeed in many cases the child may not be able to make any attempt at them, but it is equally true that it is only by their use that minor degrees of mental deficiency can be recognised.¹ Any child with less than 70 per cent. of the average intelligence, *i.e.*, a mental ratio of less than 70, is considered mentally defective.

In after-life the weakness in will-power and in self-control of these individuals is very noticeable and renders them unfit to undertake the responsibilities of life. When left to themselves they almost invariably come to grief in one way or another. If, however, they are carefully looked after from the first, and continue to be shielded from the temptations which they cannot resist, they may become, in a measure, good and useful citizens. So long as someone else is at the helm, to use Dr Tredgold's apt simile, many of them can work their passage; although, for the most part, they are quite unable to steer their own course upon life's stormy sea. When these facts are realised by the public authorities, more money will be spent in residential institutions for the feeble-minded, and much less will then be needed than is used at present for keeping them in workhouses, prisons, and Magdalene asylums. This line of campaign is certainly saner than the modern idea of sterilisation. The latter method of coping with the problem is, as already remarked (p. 823), based on entirely false premises, *viz.*, that mental deficiency is hereditary and that mentally defective children are unusually prolific.

¹ A discussion of the value and reliability of these tests will be found in "Intelligence and Disease," by Shepherd Dawson, *Med. Res. Council Spec. Report*, No. 162, 1931.

Microcephalus.

The term "microcephalus" is often applied to any child that has a small head. It is better, however, to restrict it to the small but well-defined group of cases which are sometimes called "true microcephalus." The main clinical characteristics of these children are a very small head with a narrow receding forehead, pointed vertex, and flat occiput (Figs. 30 and 31, p. 66), extremely early closure of the fontanelle, and an absence of any real paralysis. The brain is much too small, and some of its parts are out of proportion; it looks like that of one of the higher apes. It is of normal consistence and shows no areas of sclerosis, such as we find in cases of spastic diplegia from birth-injury.

Symptoms.—At birth the microcephalus may be overlooked, as the head is not relatively so small then; but it soon becomes evident, because the rapid growth, which adds 3 inches to the circumference of the normal child's cranium in the first six months, fails to take place. In well-marked cases the head measures from 14 to 16 inches in later childhood and adult life, and it is rarely more than 17 inches. In a few otherwise typical cases, however, the measurement may reach about 18 inches.

The frequent absence of the anterior fontanelle at birth, and its very early closure when present, are important points. Apart from the small forehead, the features are generally normal; the ears are large and often well formed. The palate is usually abnormally arched, and the body and limbs are apt to be undergrown, though not otherwise abnormal. In the early months the extremities may show such an exaggeration of the normal hypertonicity of infancy that spastic diplegia may be suspected. The child is generally in his third or fourth year before he can walk alone. The teeth appear at the usual times.

The degree of mental defect varies greatly—generally in direct proportion to the smallness of the head. The worst cases are quite idiotic, and most are low-grade imbeciles. A few of the milder cases, in whom the head is not very small, are little more than feeble-minded. In most of the cases speech is scanty and late in beginning, and sometimes it is never acquired. Control of the bladder and bowel is also late in

developing. Convulsions are very common—they occur in more than a third of the children.

In bad cases the infant is apathetic and unobservant in the early months, and in later childhood of a low mental type, although his movements may be very active; he is not infrequently restless, inquisitive, greedy, and cruel to other children and animals. In some of the higher grade cases, however, there may be a kindly and gentle disposition and a considerable amount of intelligence. The recurrence of fits has, of course, a very unfavourable effect on the child's disposition and on his power of benefiting from education.

The **cause** of microcephalus is unknown. The mothers are often healthy young primiparæ. Occasionally more than one case occurs in a family.

The **prognosis**, both as to length of life and mental improvement, is generally unfavourable. The children are prone to take tuberculosis and often die of it. Some of the milder cases reach adult life, and a few may be taught to earn their living under supervision.

Treatment.—While the worst cases are almost ineducable, training in an institution is desirable for the less severe. A wholesome routine of pleasant occupations and an out-of-door life do a great deal to lessen the abnormal restlessness and the tendency to convulsions.

Craniectomy was at one time recommended by some authorities. This advice was founded on a mistaken idea of the pathology of the condition, viz., that the small size of the head was due to early closure of the sutures, whereas it is because the brain does not grow that the sutures unite prematurely.

Mongolism (*Mongolian Imbecility*).

Mongolism is a common and well-defined disease which is always accompanied by mental defect. It is found in about 5 per cent. of mentally defective children of school age; but among those young children who are brought to Children's Hospitals with recognisable signs of mental defect, the proportion is much larger—probably at least 20 to 25 per cent. This disparity is partly accounted for by one of its prominent clinical features, which is a *low degree of resistance* during

infancy to most forms of infection; for something like three-quarters of these children die in the first few years of life from bronchitis, broncho-pneumonia, measles, whooping-cough, influenza, or some other ailment. The name "Mongolian Imbecility" was first given to this condition because the children's faces have generally a curious resemblance to those of the Mongolian races.¹

Although mongolism is classed with simple primary amentia and microcephalus, because like them it presents an arrest of development of the primary neurons, this condition differs essentially from the other two groups in one important respect. In the typical members of both of them the chief morbid condition is evidently a brain-lesion, which has only a slight secondary effect, if any, on the rest of the body. In mongolism, however, the morbid condition is altogether general in its distribution. The brain is not like that in normal children, but the other parts of the child's body are quite as peculiar as the brain.

Symptoms.—The main physical characteristic of mongolism consists then in the peculiar conformation of the whole body. While this is so, the special form of the face is the clinical feature which is most easily recognised (Figs. 240 to 246); indeed there is usually no difficulty in recognising it even at the time of birth.

The child's features are small and rounded, and, like the other extremities, reddish in tint. The cranium is small, round (brachycephalic), and strikingly devoid of eminences. The eyes are often too near one another, and in most cases the axes of the palpebral fissures are abnormally oblique—the outer canthus being higher than the inner. In a large proportion, also, there are prominent epicanthic folds of skin at the inner angles of the eyes. Nystagmus occurs at some time during infancy in about 10 per cent. of the cases. It is sometimes of the permanent conjugate type, but oftener of the temporary convergent or irregular variety (p. 677), in which case it is frequently accompanied by head nodding. A peculiar spotted form of lamellar cataract is not uncommon in older mongols.² Some degree of strabismus is also often present.

¹ J. Langdon Down, "Ethnic Classification of Idiots," *London Hosp. Rep.*, 1866, iii., 259.

² Pearce, Rankine, and Ormond, *Brit. Med. Journ.*, 1910, ii., 186.

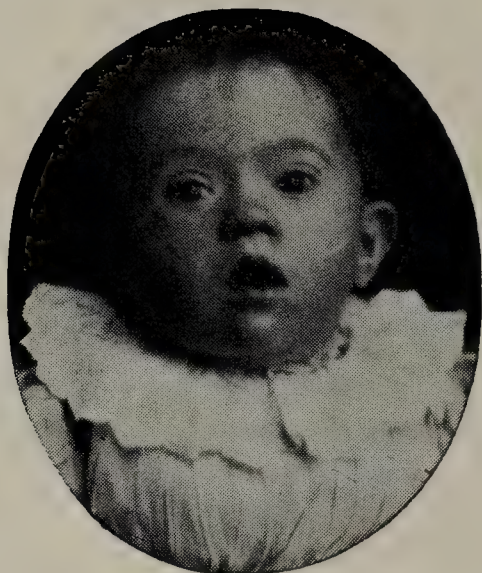


FIG. 240.—Boy of
18 months.



FIG. 241.—Girl of
2 years.



FIG. 242.—Girl of 3 months.



FIG. 243.—Girl of
 $6\frac{1}{2}$ months.



FIG. 244.—Boy of
8 months.



FIG. 245.—Mongolism. (Boy of 3 years.) Obliquity of eyes and protrusion of tongue. (Dr Melville Dunlop's case.)



FIG. 246.—Mongolism. (Girl of 9 years.) Fissuring of tongue. (Dr Melville Dunlop's case.)

The nose is snubbed and the nostrils sharply triangular in outline in many cases. They are very often narrowed owing to chronic naso-pharyngeal obstruction.

The mucous membrane of the lips is often dry and crossed by numerous vertical fissures which do not extend on to the skin like those in congenital syphilis. The teeth are generally small, and tend to become yellow and to decay early. In many cases the central incisors are rotated so that their anterior surfaces form an angle with the concavity forward.

The tongue in many cases protrudes from the mouth as if it were too big. This is due to the shortness of the oral cavity rather than to an abnormal length of the organ. During the early weeks of life its mucous membrane is quite normal in appearance. As the child grows, however, the papillæ enlarge until—between the third and ninth month—the mucous membrane has a raw and granular surface. Later—generally during the third or fourth year, but sometimes as early as the second—deep fissures form on the dorsum (Fig. 246). In more than 90 per cent. of all mongols over six years, this fissuring of the tongue is a marked symptom; and it is of special interest because, unlike the other peculiar features of the disease, it is acquired and not congenital. Although its development may be favoured by a congenital vulnerability of the mucous membrane, its main cause is certainly the persistent and forcible sucking of the tongue which these children practise.¹ The same condition will be found in any child who has practised this habit energetically for a sufficiently long time.

The ears are in most cases small, rounded, and simply convoluted (“shell-shaped”), and often project more than usual. The hair is normal in infancy, but becomes dry and scanty in older children. Alopecia areata is common, and there may be complete baldness. The skin on the limbs often shows an exaggeration of the normal mottling of childhood.

The limbs are soft and small-boned, and the joints have unusually lax ligaments, so that they can be easily hyper-extended. The shape of the extremities differs from that in cretinism. The wrist and hand are small and the latter very soft, owing to the small size of the bones and the laxity of ligaments. The fingers are rather thick for the size of the hand, but not so square-pointed as those of cretins, nor is

¹ J. Thomson, *Brit. Med. Journ.*, 1907, i., 1051.

their skin wrinkled and redundant to the same extent. The little finger is often dwarfed and shows an exaggeration of the normal curve toward the ring finger.

Malformations of various parts of the body are frequently met with—such as club-foot, cleft-palate, and hare-lip, cubitus varus, syndactyly, dwarfing of the terminal phalanges with deformed nails, hypospadias, congenital stenosis of the intestine, and others. The most remarkable fact is the extreme frequency of congenital malformation of the heart, to which attention was first drawn by A. E. Garrod.¹ Among 300 cases, of which one of us (J. T.) has notes, 47 (*i.e.*, 15·7 per cent.) showed physical signs of this. In several which were examined post-mortem, there was patency of the inter-ventricular opening, with or without other defects. As this opening should be closed by the eighth week of intra-uterine life, its persistence in many of these children seems to indicate that the cause of mongolism is one that begins to act very early in foetal life.

Delay in the growth and development of the body is also characteristic. The stature is usually stunted, so that the child looks much younger than he really is; and as he grows older the dwarfing becomes more noticeable.

The dentition is generally backward. In nearly two-thirds of the cases the first teeth do not appear until after the end of the first year, and often not till eighteen months or two years. In the rest the first few teeth come quite early, but the later ones are delayed or irregular in the time of their appearance. The fontanelle rarely closes before the third or fourth, and is sometimes still patent in the fifth or sixth year. The voluntary muscular movements are late in being acquired, so that the baby does not hold his head up until after the sixth month nor sit unsupported until the second year or later. He generally does not walk alone before the third or fourth year. The grasp is also feeble and fumbling. Speech is slow in being acquired and is guttural and indistinct.

The mongol's *disposition and character* are also peculiar to himself. Except in a few of the worst cases, they are happy, good-tempered, and very affectionate children—bright, inquisitive, and apt at mimicry. They are almost always cleanly and methodical in their ways from early infancy.

¹ "On the Association of Cardiac Malformations with other Congenital Defects," *St Bart. Hosp. Rep.*, 1894, xxx., 53.

Their *tendency to acquire infectious complaints* has been already referred to ; and they often suffer from catarrh of the mucous membranes. Blepharitis occurs sooner or later in about 90 per cent. of the cases. Bronchitis, broncho-pneumonia, and catarrh of the nose, naso-pharynx, fauces, and vulva are also common. As the naso-pharyngeal cavity is often so shallow that the vomer almost touches the back wall of the naso-pharynx, a very small amount of adenoid hypertrophy causes obstruction.

Fits sometimes occur, but not so frequently as in most other varieties of imbecility.

Diagnosis.—The distinctive physiognomy of mongolism depends on the numerous small peculiarities of conformation in the head, face, hands, and body generally. Practically, however, we diagnose the mongol mainly from his striking resemblance to other mongols, just as in other children we recognise the indications of Negro, East Indian, or Jewish parentage. They are always very like other mongols and very unlike their own brothers and sisters. The respects in which they differ from other children are comparable with, though less than, the differences between a chimpanzee and an orang-outang. In fact, they look as if they belonged to a distinct species of the human race.

The most frequent mistake by the uninitiated is to call the condition cretinism. In cretinism there is simply a coarsening of the features from the myxœdema, whereas in mongolism there is a true distortion or deformity of the features (p. 510).

Prognosis.—A large proportion of mongol babies die within the first year or two of life. In about two-thirds of these the cause of death is bronchitis with collapse, or broncho-pneumonia which is usually secondary to measles, whooping-cough, or influenza. In older children such diseases are less fatal, but many die from spinal, abdominal, or thoracic tuberculosis. In answering questions as to the child's future capabilities, it must be remembered that these children almost always improve considerably in the course of years, and sometimes make an unexpected degree of progress under careful training. From the first, however, it may be foretold that, though they will always remain imbeciles, many may in time be taught to read and write, and to conduct themselves almost like ordinary

children in their home life ; but they will never be able to earn a living. We have known one instance of a mongol girl who was married when she was twenty-eight, and had on two occasions what seemed to be a miscarriage ; her husband died within a year. So far as we know, there is no case on record of a mongol having borne a child.

Treatment.—The administration of thyroid has not in our experience been of any real value, and it often makes the children restless and nervous. Improvement often indeed takes place during its use, but it has never seemed to be any greater than it would have been without treatment.

Causation.—Considering that the condition is so common, it is curious how few definite facts we possess to help us in framing any plausible theory of its etiology. The thyroid and other endocrine glands seem normal in structure. The condition has been described as due to *an arrest or perversion of the normal development* ; and it is in keeping with this view that fully one-half of the mothers of these children are in their fortieth year or older, at the time of their birth, and that an equally large proportion of them have come at the end of large families. There are, however, many exceptions to this, for no fewer than 7 per cent. of 156 cases in which these points were ascertained were the first children of apparently healthy women under twenty-five ; and many of these mothers had several normal children afterwards. The age and state of health of the father seem to have no relation to the causation of the disease.

Congenital Syphilis in Relation to Mental Defect.

The proportion of children met with in Imbecile Institutions who show indubitable signs of congenital syphilis is usually from 1 to 2 per cent. In the practice of Children's Hospitals and Dispensaries, however, the proportion of mental cases with evidence of syphilis is much larger than in institutions. This is because most of the children die before they are old enough to be sent to institutions. In a series of 65 examples of mental deficiency, excluding spastic diplegia and mongolian idiocy, observed at R.H.S.C., Glasgow, a positive Wassermann reaction was obtained in 7·6 per cent. ; of 27 children with spastic diplegia 3·7 per cent., and of 11 mongols 9 per cent., reacted positively to Wassermann's test. These incidences of syphilitic

infection are little different from that which was found in the same class (patient of a Children's Hospital) of child generally; of 1275 medical patients, without reference to disease, 4.5 per cent. gave a positive serum reaction for lues.

Congenital syphilis may cause mental defect in many ways:—

(1) Like any other weakening influence, it may *interfere with the development of the cerebral tissues*, producing either a deformed brain or ill-grown brain cells. In recent years a number of writers have endeavoured to show, by the use of the Wassermann test, that many mentally defective children who present none of the usual signs by which congenital syphilis is recognised, nevertheless owe their condition to this cause. In a very few instances the diagnosis made in this way has been followed by improvement in the mental symptoms from the use of salvarsan or one of its equivalents.



FIG. 247.—Idiocy from Congenital Syphilitic Brain Disease. (Girl of 8½ years.)

(2) Occasionally we see children suffering from various forms of paralysis, with amentia, which are apparently due to syphilitic *endarteritis* of the cerebral vessels.

(3) *Chronic hydrocephalus* is not very uncommon in syphilitic babies, and often recovers satisfactorily under specific treatment.

(4) *Intracranial gummata* are sometimes met with, though they are very rare.

(5) Ashby has described a form of Jacksonian convulsions in babies with congenital syphilis, which depends on the occurrence of *acute meningo-encephalitis* of the cerebral cortex and ends in sclerosis and severe dementia (p. 674) (Fig. 247).

(6) The commonest and most interesting intracranial disease of specific origin in children is the chronic meningo-encephalitis, the morbid anatomy of which is similar to that found in adult

general paralysis,¹ and which gives rise to *progressive dementia* in older children.

Progressive Dementia (*Juvenile General Paralysis*).—This condition usually begins insidiously. Its first symptom may appear as early as the fourth year, or even before that age, but most of the patients are at least six or eight years old; and girls are more often affected than boys. The disease may set in in children who had previously seemed quite normal; but, in most cases, indications of mental defect have been present from early infancy.

Symptoms.—When this disease begins in childhood its main bodily symptoms are those of slowly advancing spastic diplegia, while the mental condition is one of simple steadily progressing deterioration. Grandiose ideas, so characteristic of the disease in the adult, are hardly ever met with in the child. Along with the paralysis and dementia there is usually increasing debility with spastic diplegia, and there may be severe pains in the back and limbs; often, also, there are recurrent convulsive seizures (“congestive attacks”).

In older children (from eight to twelve) we may also meet with this simple type, and it is well to remember that cases of acquired spastic diplegia and progressive dementia in children of school age are often due to this cause. Until a decade ago syphilis was the commonest cause of acquired mental deficiency. Since then, however, epidemic encephalitis is the most frequent etiological factor (p. 770).

The more typical cases of juvenile general paralysis are found in older children and adolescents. In these the early symptoms often consist in attacks of severe headache with vomiting, or in ill-defined epileptiform congestive attacks. Sometimes there is only a passing loss of consciousness or of speech. Later, eye symptoms develop. There may be irregularity in the shape and size of the pupils and loss of the light reflex (Argyll Robertson pupil); optic atrophy sometimes occurs. The knee-jerks are greatly exaggerated and in some cases ultimately lost, and the plantar reflex gives an extensor response. Facial irritability is sometimes present in an exaggerated degree. In some cases a chorea-like tremor is

¹ Thomson and Dawson, *Lancet*, 16th Feb. 1895, 397; Thomson and Welsh, *Brit. Med. Journ.*, 1st April 1899, 784.

one of the early symptoms, and the articulation may also be characteristically affected.

The mental deterioration is profound and steadily progressive; and the patient becomes more and more like a baby in his ways and loses memory, interest in outside things, and natural affection. If there are younger children in the house, he may be very jealous and attack them. Although hallucinations are apparently absent in the younger cases, there are sometimes, in the older children, a few of an elementary and childish sort. The defective speech and other symptoms get worse and worse, and the child becomes extremely emaciated and finally paralysed. The limbs sometimes develop severe contractures.

The duration of the case, if no intercurrent disease sets in, is very protracted, sometimes lasting about two years, generally three or four, and occasionally as long as six or seven. In the older cases puberty is either late or it does not appear at all.

Treatment of juvenile general paralysis is less satisfactory in the child than in the adult, probably because it has been longer in existence before being recognised. Arsenical treatment is usually very disappointing, but the condition may be arrested, especially if the case is got early, by infection with malaria.

Tay-Sachs' Disease.

For this malady there have been numerous synonyms suggested (*amaurotic family idiocy, amaurotic family dementia, infantile cerebral degeneration, agenesis corticalis* or *arrested cerebral development, familial macular degeneration, and familial cerebral degeneration*) with the intention of providing a term descriptive of the various manifestations encountered, but not one is sufficiently all-embracing to include all varieties of the disease. When one appreciates that the disease may make its appearance at any age during childhood, that there may be only one example in a family, and that macular changes may or may not be present, such descriptive adjectives as "infantile," "familial," and "macular" would seem hardly appropriate. True, in all histological examinations of the brain, degeneration of nerve cells has been encountered, and, as it occurs very frequently in several members of a family, familial

cerebral degeneration would be the best alternative. The use of the designation, Tay-Sachs' Disease, has, however, the advantage that it does not bind us to any one view regarding the classification or etiology, and keeps alive the memory of the two men who first described it.

From a brief account of the salient facts in the history of the disease the best idea of this clinical entity is obtained. It was in 1881 that Waren Tay¹ of London recorded the case of a child of twelve months with generalised paresis and impaired mental development, and in whom ophthalmoscopic examination revealed a whitish area with central red spot at the macula. Tay was able subsequently to relate that three similar cases occurred in the same family and that all the children died before they reached the age of two years. In 1887 Sachs,² unaware of Tay's writings, published an account of the clinical appearance and pathological findings in the case of a child with idiocy and blindness which agreed in its details with that described by Tay. At this time the child was the only member of the family affected, but within a period of six or seven years Sachs had observed a subsequent child in the family become similarly affected and, in addition to several isolated examples, another family in which four children all became affected apparently with a similar disease and died. In 1896 Sachs³ was able to collect 19 cases from the literature and to corroborate Carter's observation that the disease was limited to the Hebrew race. Later experience, however, has shown that this racial limitation is not absolute. Thomson⁴ records the condition in two infants of purely Scottish parentage.

In 1903, F. E. Batten,⁵ under the title "cerebral degeneration with symmetrical changes in the maculæ in two members of a family," described the cases of two brothers who developed mental deficiency with macular changes at the age of four to six years. These patients were not Hebrews. In 1914 Batten⁶

¹ Waren Tay, *Trans. Ophth. Soc., U.K.*, 1881, i., 55.

² B. Sachs, *Journ. Nerve and Ment. Dis.*, 1887, xiv., 541.

³ Sachs, *New York Med. Journ.*, 1895, lxiii., 697.

⁴ J. Thomson, *Clinical Study and Treatment of Sick Children*, 4th ed., 1925, p. 714.

⁵ F. E. Batten, *Trans. Ophth. Soc., U.K.*, 1903, xxiii., 386.

⁶ F. E. Batten, *Quart. Journ. Med.*, 1914, vii., 444.

described another family in which three of the children developed convulsions with progressive mental degeneration and spastic paralysis at the age of three and a half years, and in one of whom there was in addition optic atrophy with a patch of pigment at the macula. All died before reaching the age of six years. Other examples of mental symptoms with eye changes have been described singly or in family groups, and are frequently spoken of as the juvenile type of Tay-Sachs' Disease or amaurotic family idiocy.

Until 1922 the recorded examples had occurred either during infancy or after three and a half years, but in that year Torrance¹ put on record a family of three children, each of whom developed nystagmus with mental deterioration and fundal changes soon after the age of one year, and who all died as complete imbeciles before they had reached the age of four years.

Although there are decided differences in these various groups of cases, as well as others recorded in the literature, as regards age of onset, race, presence or absence and degree of mental changes, and presence or absence as well as type of fundal changes, the whole series form a sufficiently homogeneous group to warrant the assumption that they represent one clinical entity. The gross appearances of the brain vary from atrophy of the cerebrum or cerebellum to a brain that looks normal on naked eye examination, but the minute anatomy of which reveals in all the cases examined the one fundamental change, viz., a degeneration of the nerve cells, at times limited to one portion—cerebrum or cerebellum—but at other times involving the whole central nervous system (cerebrum, cerebellum, and cord). The earliest change is one of swelling of the nerve cell and fibrillar processes, with later chromatolysis and disintegration of the dendrites and cells until they completely disappear. Curiously, the axis-cylinders remain intact. There is no appearance of an inflammatory process.

It is generally held that the whole process is of the nature of a degeneration, premature because of some congenital inherent weakness in the nervous system. Consanguinity, nervous disease in the ascendants, or syphilis do not seem to play any part in the etiology.

The *symptomatology* of the condition is more or less alike,

¹ H. W. Torrance, *Glasg. Med. Journ.*, 1922, xcvii., 193, 263, 341.

no matter the age of the patient. In all the patient is born apparently normal and remains so for a variable time, but this period would seem to be more or less constant for each family as all siblings are affected at practically the same age. In the *infantile type* the child, almost invariably a Hebrew, remains apparently normal for some months, when he ceases to develop instead of showing the normal progress. He becomes listless, ceases to take an interest in his surroundings, and ultimately becomes blind. The muscular weakness proceeds to complete paralysis, which may be either spastic or flaccid in nature, with increased or absent reflexes (Fig. 248). There may be nystagmus or convulsions. Ophthalmoscopic examination will reveal the characteristic cherry-red spot and optic atrophy.

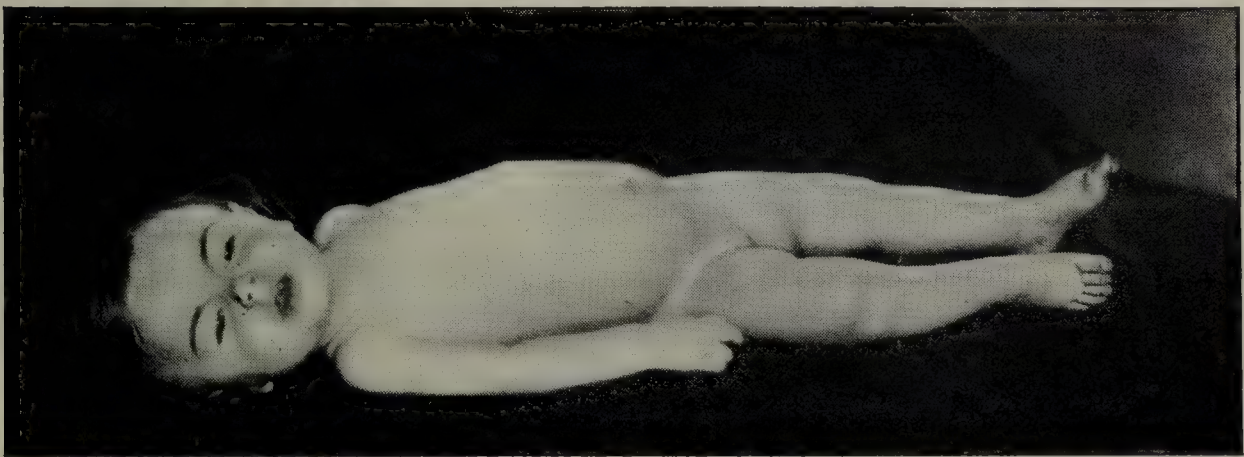


FIG. 248.—Amaurotic Family Idiocy. Late stage. (Dr W. B. Drummond's case.)

The child steadily emaciates, and is liable to develop some pulmonary or gastro-intestinal complication which may hasten the invariable fatal termination.

In the *juvenile type* of the disease the child, who in contrast to the infantile variety is usually but not invariably a Gentile, retains his normal mental and physical health for a period varying between one and fourteen years before the mischief declares itself. The first manifestation may be a convulsion, nystagmus, or simply a cessation of the normal progress. It is interesting to note that in the case of siblings not only is the age of onset the same but the type of onset is identical. Whatever the nature of the first symptoms, sooner or later there sets in mental deterioration along with muscular weakness. These proceed at a variable rate and assume a variable degree. In many cases the child ultimately becomes a complete imbecile, unable to rise out of bed, taking no interest in his surroundings, and possibly totally blind. The limbs may be spastic or flaccid,

or spasticity and flaccidity may alternate, and the reflexes may be normal or exaggerated. Examination of the fundi will reveal optic nerve atrophy and possibly some macular change closely simulating that found in the infantile variety. A cherry-red spot has been described, but more frequently a zone of pallor or a mass of pigment is the abnormal feature; in some cases masses of pigment may be apparent in the periphery and



FIG. 249.—Idiocy from Infantile Cerebral Paralysis. (Girl of 14 months.)

in others again no retinal abnormality has been discovered. In the siblings affected the fundal changes are most variable so far as presence and nature are concerned.

The disease, as already mentioned, invariably proceeds to death, and the earlier the age of onset the more rapidly. No method of arresting it is known.

Amentia due to Gross Cerebral Lesions.

In any form of paralysis from cerebral disease or injury there may be some degree of mental impairment.

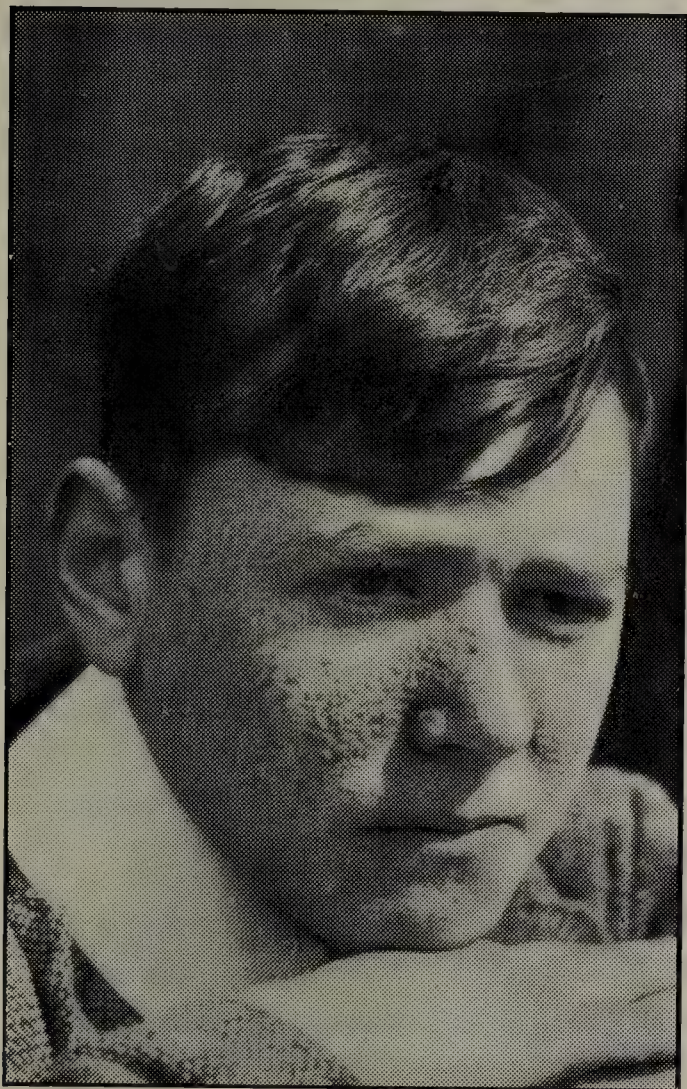
In cases of cerebral diplegia (Fig. 249) and hemiplegia, from meningeal hæmorrhage at birth or from injury, in poli-encephalitis, and in chronic hydrocephalus following meningitis, the damage to the intellect may be very severe or comparatively slight, according to the parts of the brain damaged (Chapter XXIX., p. 744). Many slightly paralysed children who are by no means imbeciles have a slight degree of mental instability and emotional weakness, or a tendency to epileptic convulsions.

In cases of spastic diplegia the child has often considerable mental capacity and fairly good judgment. Occasionally, even when the limbs are very spastic and choreic, a surprising amount of manual education is possible. Some of these children develop great powers of perseverance, and may in

time be taught to do quite difficult things—though, of course, never very deftly or gracefully.

Tuberous Sclerosis.

Tuberous sclerosis is a developmental disease of unknown causation, which is characterised clinically by convulsions and mental deterioration, and anatomically by numerous small



FIGS. 250 and 251.—Adenoma Sebaceum in Tuberous Sclerosis.
(Dr R. D. Clarkson's cases.)

rounded areas of sclerosis in the brain, and sometimes by a peculiar eruption in the skin of the face (adenoma sebaceum) (Figs. 250 and 251), and congenital tumours in various of the internal organs. The condition was first described by Bourneville in 1880.¹

The main *symptom* of the disease is the occurrence of general convulsions. These are apt to be numerous and

¹ Bourneville, *Arch. de Neurology*, 1880-81, i., 69 ; L. Findlay, *Review of Neurology and Psychiatry*, 1905, iii., 391 ; J. S. Fowler and Carnegie Dickson, *Quart. Journ. of Med.*, 1910, iv., 43.

severe, but apparently have no other peculiar characters. They often begin in early infancy, but are sometimes delayed until later childhood. Severe mental defect is generally present from the first; but, in some cases, the intellect only begins to suffer after the convulsions have lasted for years. The other manifestations of the disease are, as already mentioned, the presence of adenoma sebaceum (an eruption of little red nodules on the skin of the nose and cheeks), and isolated undiagnosable tumours elsewhere which usually contain embryonic elements. Such tumours have been described in the heart (rhabdomyomata), kidneys, thyroid, thymus, pancreas, mammæ, and duodenum. Spastic paralysis and contractures occasionally occur, and congenital malformations of the heart and of the central nervous system, such as spina bifida, have been described. When the skin is not affected, the *diagnosis* of tuberous sclerosis is merely a matter of conjecture; but probably all mentally defective children with epileptiform convulsions who have adenoma sebaceum are instances of this disease.

No *treatment* is of any avail; and the patients die either during childhood or in adult life.

Relation of Epilepsy to Amentia.

True epilepsy, when it lasts for any time, nearly always has a serious effect on the mental condition in childhood.

Although history relates that such prominent and remarkable men as Julius Cæsar, Mahomet, and Napoleon were so affected, an entirely sane epileptic child is rarely seen. Shanahan¹ found only 10 per cent. of the epileptics in Craig Colony, New York, normal; he considered, however, that only 15 per cent. had deteriorated from normality, since 75 per cent. had been defective from birth. Lowenstein,² on the other hand, only found six out of sixteen examples of idiopathic epilepsy abnormal. Dawson and Conn³ found the mental ratio of typical epileptic children who passed through the wards of R.H.S.C., Glasgow, varying between 117 and 49: in eleven children the ratio was less than 70, *i.e.*, the children

¹ W. T. Shanahan, *Med. Journ. New York*, 1920, ii., 884.

² P. S. Lowenstein, *Amer. Journ. Med. Sci.*, 1922, clxiii., 120.

³ S. Dawson and J. C. M. Conn., *Arch. Dis. Child.*, 1929, iv., 142.

were definitely mentally defective. These authors found no correlation between the duration of the illness, the severity and frequency of the fits, and the mental deterioration, but, as previously mentioned (p. 709), when the fits ceased there often took place an improvement in the mental condition.

As the fits continue, the patient's character and disposition alter and he tends to lose his natural shyness, or he becomes moody, impulsive, passionate, and full of reckless mischief. Occasionally maniacal symptoms appear.

Epileptiform fits are common in all the usual varieties of amentia except cretinism and mongolism, in which they only rarely occur. They are especially frequent in cases with gross naked-eye changes in the brain; and their frequency varies in direct proportion to the severity of the mental defect. In the large majority of cases of amentia with convulsions the fits are due to the cerebral lesion which is causing the mental condition. In comparatively few is the intellectual weakness altogether secondary to the fits. In all cases, however, the occurrence of convulsions has a serious effect on the mental state, which deteriorates so long as they continue to recur, and ceases to do so when they stop. Later, the excitement caused by the fits diminishes and the condition passes gradually into one of dementia.

In regard to the child's education, the recurrence of epileptiform convulsions is always important; for even when the mental defect seems slight, the continuance of the epilepsy prevents all hope of permanent mental improvement by education. Some of the children show considerable intelligence at first; but, as the disease progresses, they forget what they have learned, so that their schooling is apt to be only a cause of worry and excitement to them. When such children are sent to an institution where they are not troubled with much brain work, the fits often become less frequent or cease for the time, owing to the healthy influence of the regulated open-air exercise, comparative freedom from restraint, and suitable occupations.

Moral Imbecility.

There can be no doubt that cases of grave moral defect with average intellectual capacity are occasionally met with after puberty which are apparently of congenital or developmental

origin.¹ These cases are to be classified as moral imbeciles, according to the Act, for, although their *intellectual* powers are normal, they are permanently lacking in other even more important forms of mental capacity—moral sense and wisdom—and experience shows that their immoral conduct is not influenced by the treatment which is suitable in similar cases in normal people, and also that it continues during life. Such cases, however, need not be considered here, because although their defect is probably of congenital origin, it scarcely ever shows itself during childhood; and, when it does, it is not possible at that stage to distinguish its manifestations from those of mere childish naughtiness.

In later childhood we sometimes meet with individuals who do their lessons well enough, but have a very poor perception of right and wrong, like that of a much younger child. Many of these cases seem to depend mainly on a *delayed development* of the moral sense; and, if they are placed in a favourable environment and judiciously managed, they recover entirely in the course of time and learn to behave like normal people. They are not to be regarded as moral imbeciles, for their shortcomings are merely due to retardation and not to permanent defect of their moral development.

Moral defects of many kinds are very common among higher-grade mental defectives; and when they form the most prominent feature in the case, the patient may be certified as a moral imbecile; and this is very generally done. In many cases so certified, however, the after-history shows that the moral offences soon lessen and often cease altogether under judicious treatment although the intellectual defect persists. It therefore seems very probable that these children are not really moral imbeciles at all. They are merely ordinary mental defectives whose childish naughtiness has been morbidly exaggerated till it has got quite out of hand, partly on account of their want of sense and self-control, but largely because they have had no proper home training during their early years.

The home treatment of older mentally defective children who show much moral perversity is extremely difficult, if not impossible. When removed to a well-ordered institution, however, they often improve satisfactorily and learn to behave well.

¹ A. F. Tredgold, *Mental Deficiency*, 3rd edition, London, 1920, 348.

CHAPTER XXXIII

DEFECTS AND DISEASES OF THE MUSCLES

Congenital Pectoral Defect

THE commonest form of congenital absence of muscle met with in practice is that found in the deformity known as "congenital pectoral defect." It is always unilateral, and affects not only



FIG. 252.—Congenital Pectoral Defect, with absence of Nipple on left side.

the muscles but also all the various structures which normally occupy the pectoral region and its neighbourhood. The greater and lesser pectoral muscles are either quite absent or partially so, and the subcutaneous fat over them is scanty compared with that on the normal side. The mamma is either absent or very small; and occasionally, as in Fig. 252, there may be no trace

of a nipple. In fully a quarter of the cases¹ there is also a defect in the wall of the chest which involves some of the ribs and cartilages (Fig. 253), and sometimes also the adjacent margin of the sternum. The defect is generally found somewhere between the second and fifth ribs. Its size and shape vary in different cases, but it always occupies an area which might be covered *in utero* by the child's fist or forearm when the

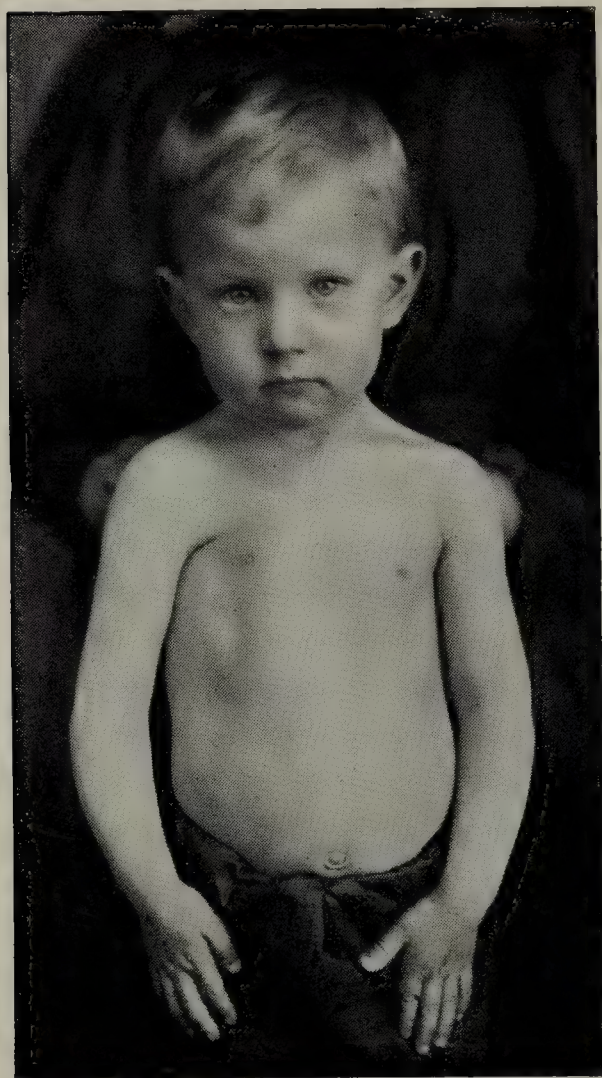


FIG. 253.—Congenital Defect of Pectoral Muscles and Chest Wall. (Boy of 5 years.)

elbow was flexed. It is interesting in this connection to note that in about one in seven of the reported cases there was also some deformity of the hand or forearm of the affected side. This almost always takes the form of dwarfing of the hand and fingers with webbing of the latter.

The *pathology* of congenital pectoral defect is obscure. The lesions correspond to nothing found in the normal course of development, nor can they be attributed to any central lesion of the nervous system. It seems almost certain that this defect, like congenital elevation of the scapula, with which it is occasionally associated, is caused in some obscure way by pressure exerted *in utero*.

The *diagnosis* presents no difficulty to anyone acquainted with the condition.

The *prognosis* as to usefulness of the arm on the side affected is good, provided the hand is normal. The defect of the pectorals interferes very little with the patient's capacity for ordinary work. No treatment is indicated.

¹ J. Thomson, *Teratologia, Quarterly Journal of Antenatal Pathology*, Edin., 1895, ii., 1.

Congenital Diaphragmatic Hernia.¹

Hernia into the thoracic cavity through a congenital defect in the diaphragm, of the stomach, colon, or small intestine, sometimes along with other organs such as the spleen, liver, and pancreas, is occasionally met with, and gives rise to symptoms which are very perplexing if the possibility of its occurrence is not remembered. The gap in the diaphragm is generally of considerable size and occurs much more frequently on the left than on the right side.

Symptoms.—There may be an entire absence of symptoms at first; but sooner or later, sometimes not for years, attacks of interference with the respiration and circulation occur. Often, also, there are symptoms and physical signs of a kind which cannot be explained by any other condition. There may be dyspnœa which varies greatly from time to time, with deficient expansion of the lower parts of the chest, or displacement of the heart with cyanosis. The affected side of the thorax moves less than the other, and shows an absence of breath-sounds; it sometimes gives a tympanitic, and sometimes a dull note, according to the organs involved in the hernia. A short time after a severe attack of dyspnœa the patient may be quite comfortable, and little or nothing amiss may be found on physical examination. If the bowel becomes compressed, however, vomiting and other indications of intestinal obstruction rapidly set in.

In older children, if the hernia is not a large one, there may only be dyspnœa, a feeling of fullness, and cyanosis, after meals; and these patients may live a long time with comparatively little discomfort.

Diagnosis.—Although a precise diagnosis may not be possible at first, diaphragmatic hernia should always be suspected in dextro-cardia, and whenever symptoms such as those described above occur with greatly varying intensity, especially in young infants. X-ray examination after a barium meal will reveal the state of matters (Figs. 254 to 257).

Treatment.—When acute strangulation takes place an operation should be attempted for its relief; but, under other circumstances, surgical proceedings are inadvisable.

¹ E. B. Leech and C. H. S. Redmond, *Med. Chronicle*, April 1909, l., 1.

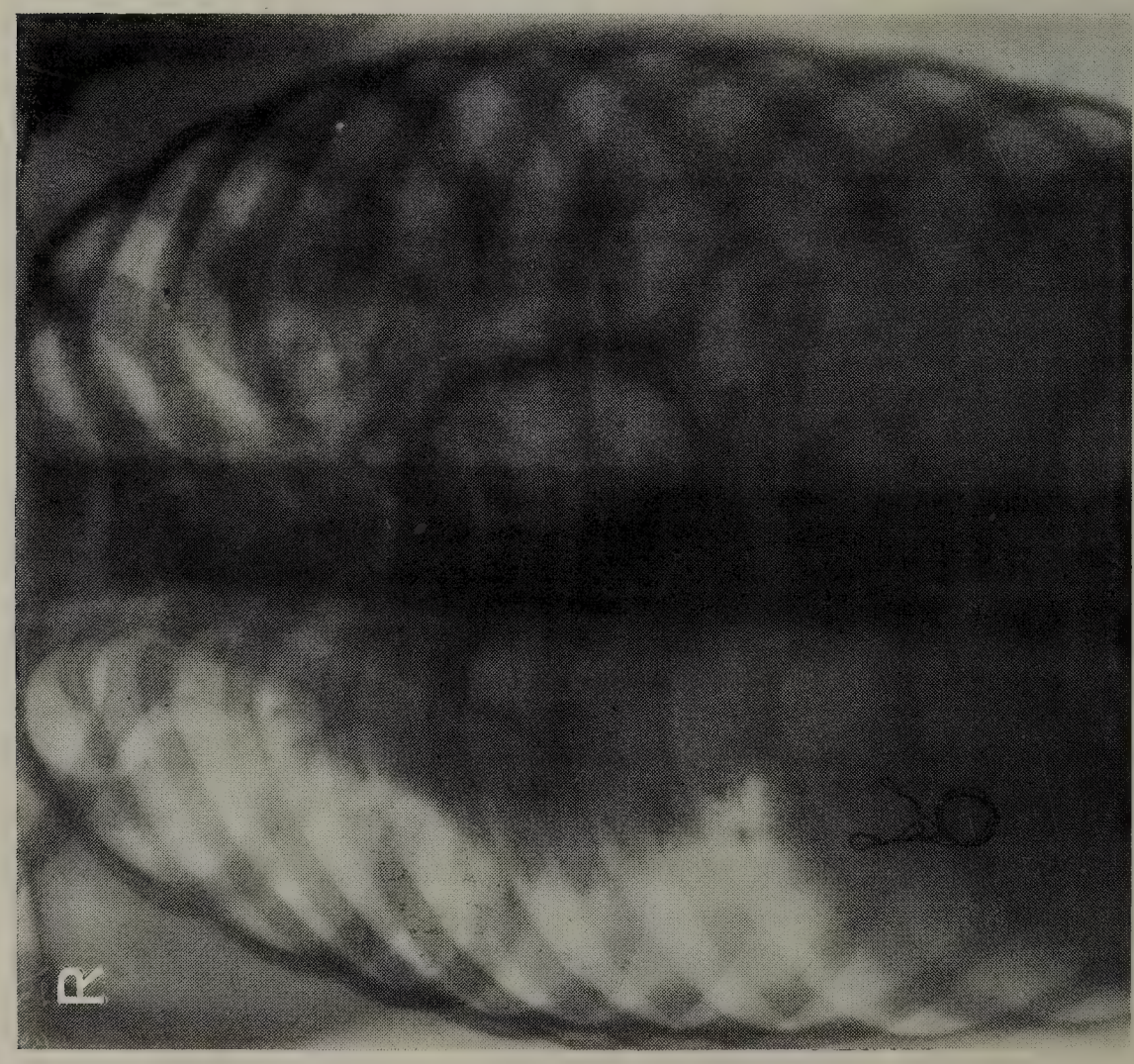


FIG. 254.—Skiagram in case of Congenital Diaphragmatic Hernia. (Girl aged 11 years.) Note dextro-cardia, air-filled cavity (stomach) in normal cardiac area, and mottling throughout left thorax due to hernia of small intestine.

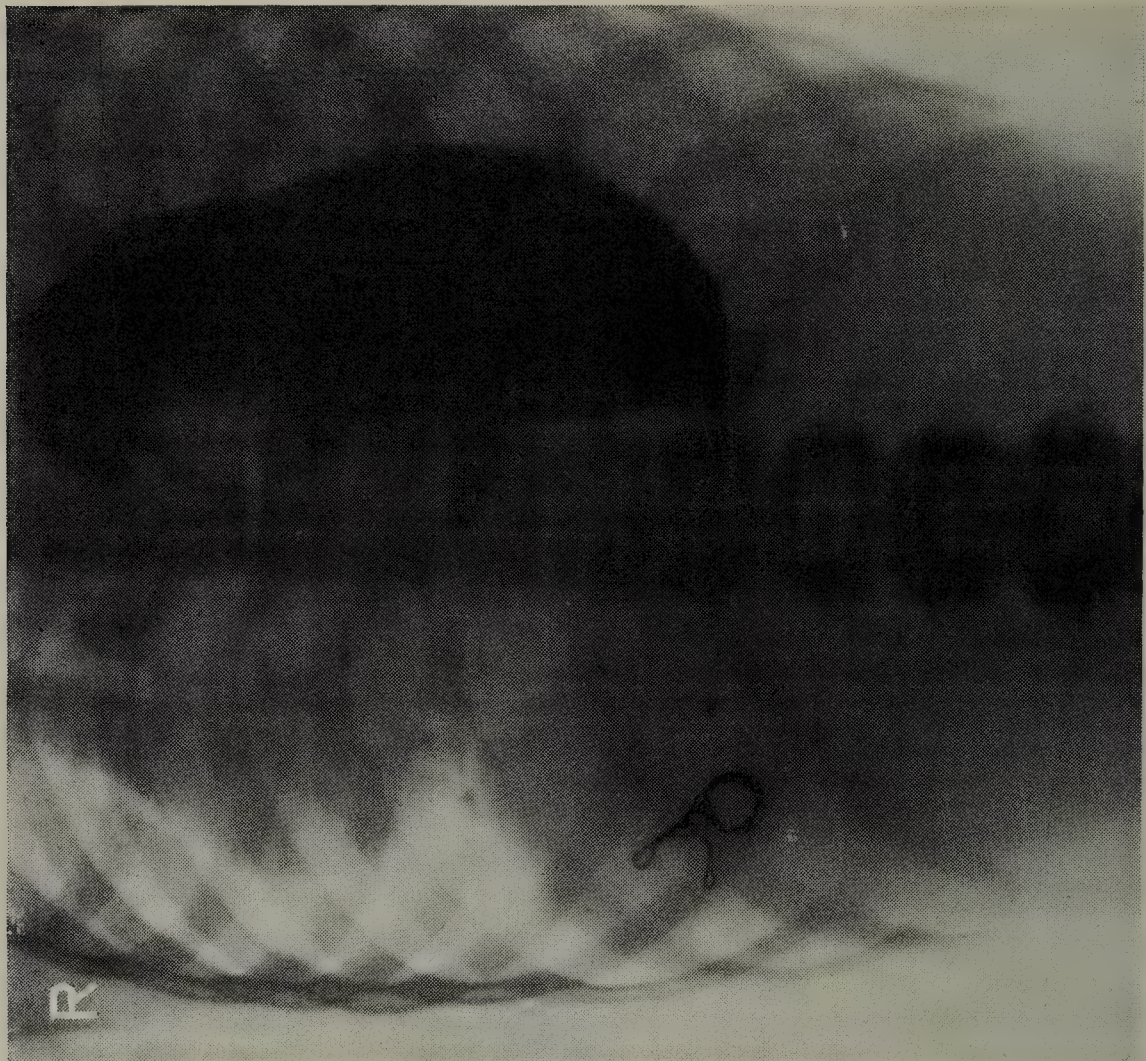


FIG. 255.—Same case as Fig. 254. Skiagram taken immediately after a barium meal. Note stomach in left thorax filled with opaque substance.

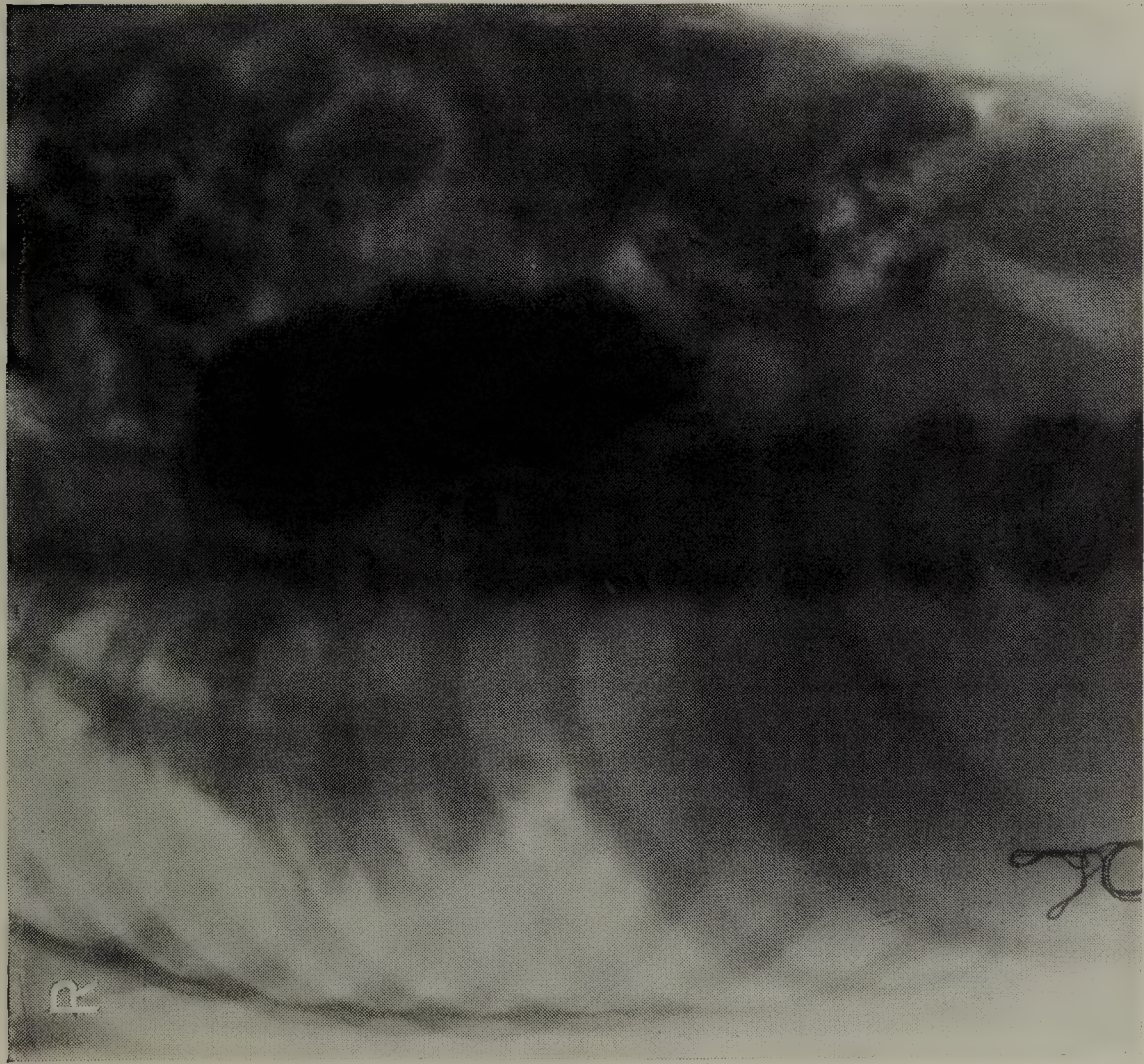


FIG. 256.—Same case as Fig. 254. Skiagram taken three hours after a barium meal. Note diminution in size of stomach shadow and presence of the opaque substance in small intestine.

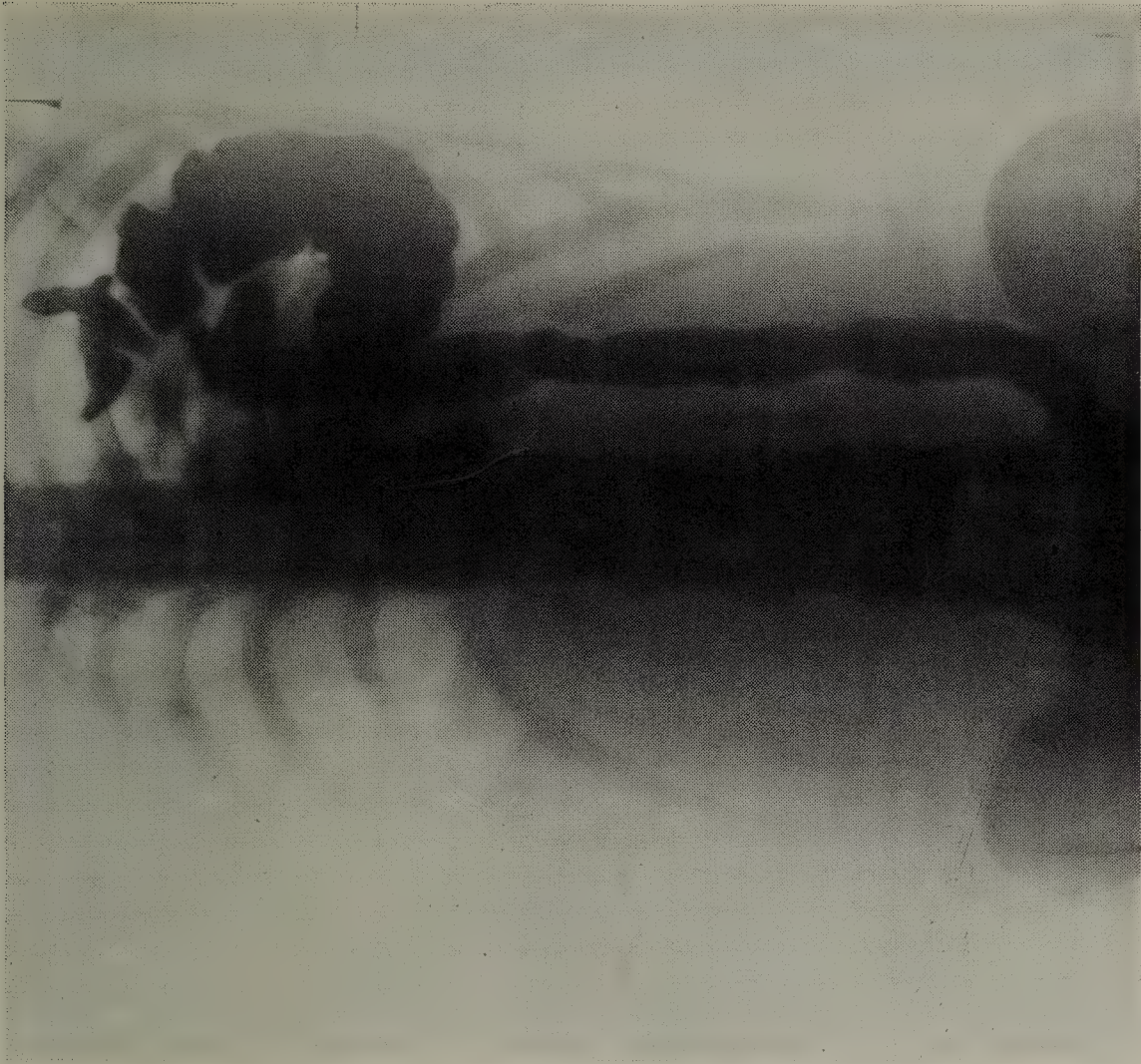


FIG. 257.—Same case as Fig. 254. Skiagram taken twelve hours after barium meal. Note situation of colon entirely on left side.

Congenital Defect of the Abdominal Muscles.

Congenital defect of the abdominal muscles is a very rare but extremely interesting abnormality both from a clinical and pathological point of view.¹

Clinical Features and Morbid Anatomy.—All the cases reported have occurred in boys. About half of the patients died when only a few months old, any respiratory disease being rapidly fatal. One of them² was living at six, and another³ at seventeen years. In Osler's case there was frequent micturition and slight polyuria; in none of the others were any urinary symptoms recorded.

The thorax is fairly normal in shape at the time of birth; but, when respiration begins, a pigeon-breast rapidly develops owing to there being no abdominal muscles to moor the lower margin of the chest-wall to the pelvis. The deep furrowing of the flaccid abdominal wall is very striking (Figs. 258 and 259). On section it is found that the only muscular tissue left consists of portions of the oblique and transversalis muscles, just below the costal margin and at the upper part of the abdomen behind; but over most of the abdominal surface there is nothing left but skin and fatty tissue. The aponeuroses are absent. The only muscles which show any defect are those of the abdominal wall.

The bladder is much enlarged—about the size of a hen's egg—and its muscular wall greatly thickened (Fig. 260), but no obstruction to the outflow of urine is present. The urachus is very short. Both ureters are dilated and tortuous, so that they look like pieces of bowel (Fig. 260). There is a moderate degree of hydronephrosis. The testicles are situated high up in the abdominal cavity.

Causation.—The best explanation of the origin of the defect is that given by Stumme. According to his theory the muscles have been prevented developing, during intra-uterine life, by the prolonged pressure exerted outwards on the abdominal wall by an enormous distension of the bladder. This takes place during the early period of pregnancy. Long before the

¹ A description of a typical case of this abnormality, with an account of the literature of the subject, will be found in a paper by Lewis Thatcher in the *Edin. Med. Journ.*, N.S., 1913, xi., 127.

² W. Osler, *Johns Hopkins Hosp. Bull.*, 1901, xii., 331.

³ Stumme, *Mitth. a. d. Grenzgeb., d. Med. u. Chir.*, 1903, xl., 548.



FIG. 260.—Post-mortem appearances.



FIG. 259.—After death.



FIG. 258.—During life.

child is born the bladder has become greatly hypertrophied and the organ has contracted so as no longer to press on the abdominal wall. The clinical appearances and the facts of the morbid anatomy seem to support this hypothesis. The wrinkled state of the surface of the abdomen is in keeping with the idea that it has been much over-distended; and the portions of the abdominal wall which are found still to contain muscular tissue are so situated that even an enormously distended bladder could not have exerted much pressure upon them. The explanation of the distension and thickening of the bladder wall is probably the same as in other cases of congenital hypertrophy of the organ (p. 449).

Myositis Ossificans.¹

Progressive multiple myositis ossificans is a very rare disease of childhood, in the course of which portions of many of the muscles undergo a bony transformation. The muscles which are chiefly affected are those of the scalp, neck, trunk, and limbs, and the masseters; while the disease leaves unaffected those of the hands and feet, the muscles of expression, those of the eye and ear, the diaphragm, the muscles of the pelvis and genital organs, the sphincters and all the unstriated muscles. The disease comes on in short attacks which usually run their course in from two to six weeks, but in the later stages the free intervals may last for months or even for years. The first appearance of the muscular affection often begins in infancy (in several reported cases at five months old), though it may not appear till later childhood. The part first attacked is generally the scalp or neck, or part of the back; later other parts of the back, the loins, the axillary folds, and the muscles of the upper arms and thighs are those chiefly involved.

Spontaneously, or possibly after some slight injury, a well-defined rounded or oval swelling appears over some portion of muscle (Fig. 261). It is doughy and elastic to the touch, sometimes slightly œdematous, and there is little or no tenderness. The skin and subcutaneous tissue seem to be involved

¹ Lydia M. de Witt, *Amer. Journ. Med. Sci.*, Sept. 1900, cxx., 294; H. D. Rolleston, *Clin. Journ.*, 23rd Jan. 1901, xvii., 209; A. E. Garrod, *St Bart. Hosp. Rep.*, 1907, xliii., 43.

as well as the muscle. There may be a slight rise of temperature. After the swelling has gone on increasing in size for some days, it gradually diminishes; and, as it is subsiding, a small angular mass of bony hardness is generally felt. When this occurs, it remains permanently, and usually increases in size every time the same part is affected. Some of the

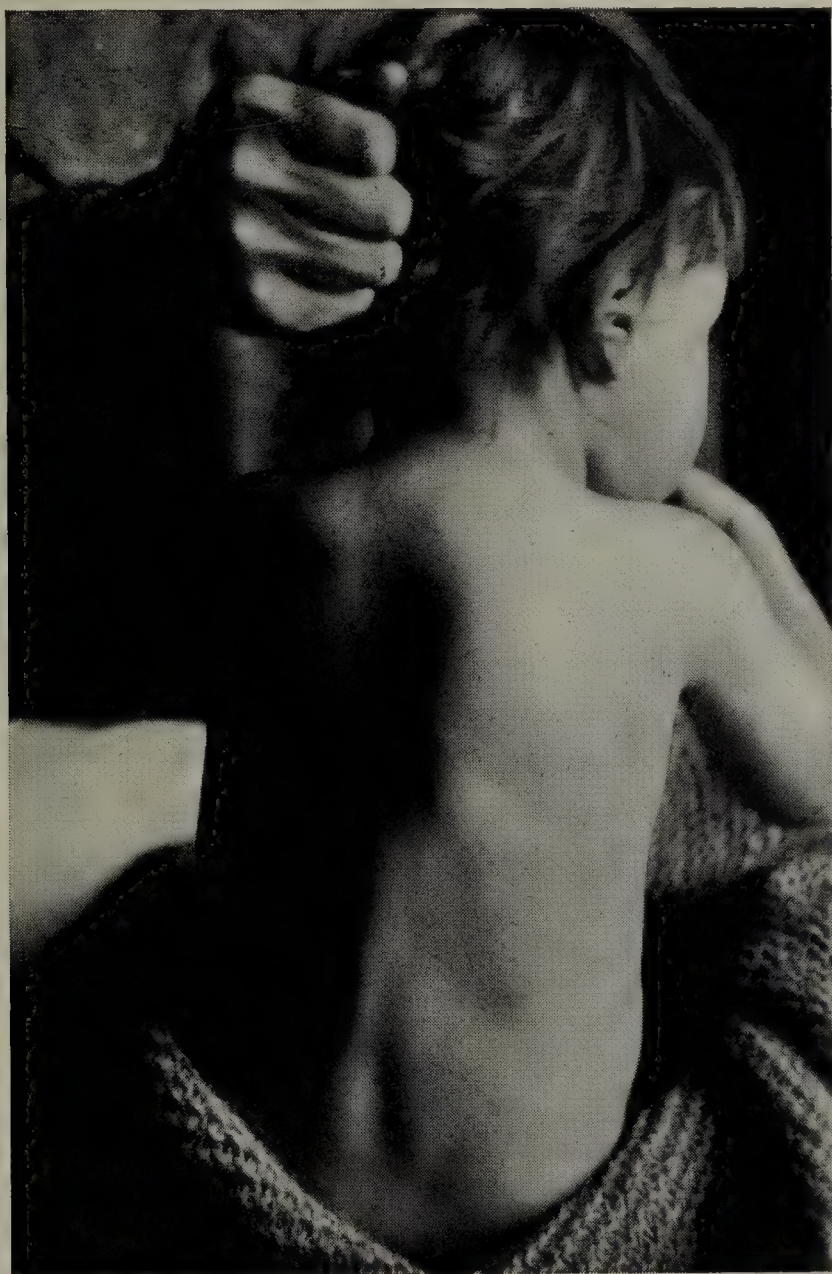


FIG. 261.—Myositis Ossificans. (Girl of 3 years.)

swellings during infancy leave no palpable hardness behind them. During the attack, as well as in the intervals, the child seems quite well. The distribution of the swellings is generally more or less asymmetrical. There are no sensory changes except slight tenderness; and the intellect is never affected.

As the disease progresses walking becomes difficult, but it is a long time usually before the child's school attendance is interfered with. The spread of the disease in the muscles of the neck, chest, and back, however, gradually weakens the

power of the respiration, so that any chest affection is apt to be dangerous. The involvement of the masseters, which may occur in the later stages of the disease, may seriously interfere with the child's feeding by preventing opening of the mouth. The disease often becomes more active about the time of puberty. In the girl whose photographs are here reproduced, fresh swellings continued to form at varying intervals until

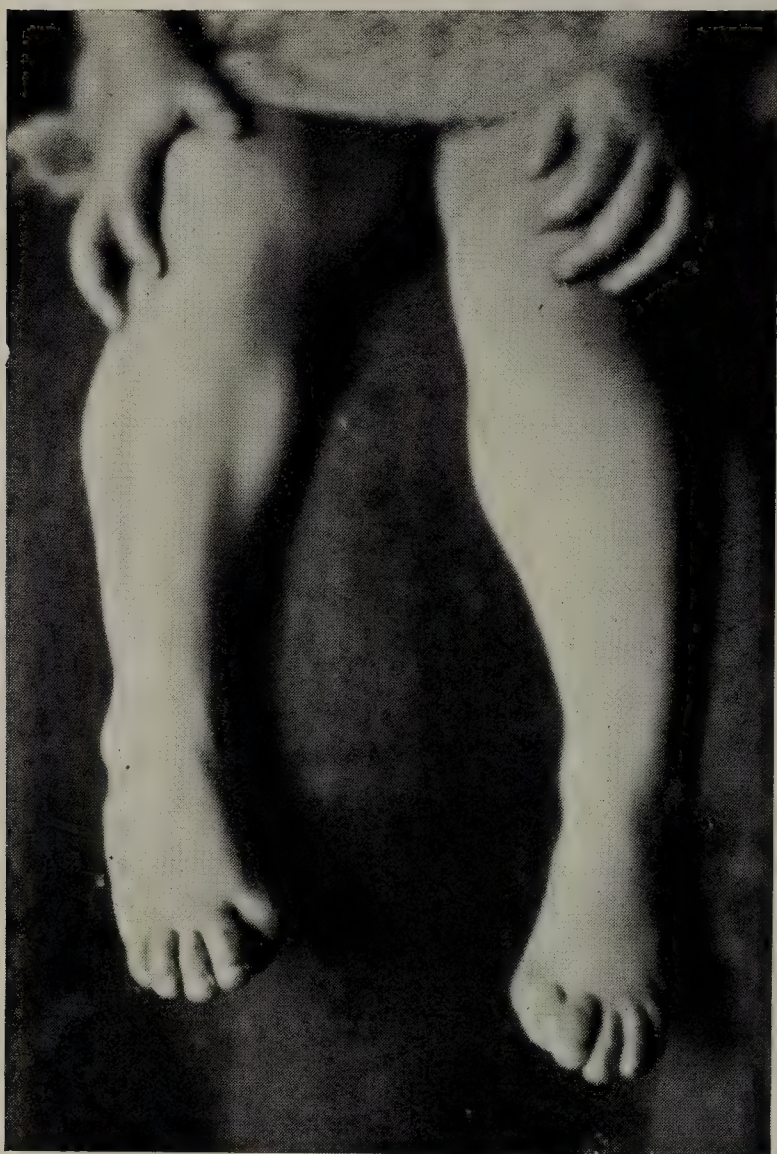


FIG. 262.—Myositis Ossificans. (Girl of 3 years.)
Deformity of thumbs and great toes.

she was nearly eleven years old, but none have appeared since. At the age of twenty-one, though much crippled by the stiffness of her limbs and body, she enjoyed fairly good health and did a surprising amount of housework.

It is a peculiar and most interesting fact that about three-quarters of the cases show, from birth, a special form of *microdactyly* of the thumbs and great toes (Fig. 262), which seems to be pathognomonic, and proves that, although the muscular lesion is not present at birth, the tendency to it is

congenital. The thumbs are small and short, usually with an apparent partial ankylosis of the joints, which are limited in their movements. The great toes are dwarfed and slope outwards (*hallux valgus*), so that the feet look like those of a woman who has been wearing short and pointed boots. Their joints also may be defective. In some cases exostoses form on the digits, on the cranium, or elsewhere.

The cause of the disease is absolutely unknown, and no instance of its hereditary or family occurrence has been reported. It is about three times as common in boys as in girls.

No treatment has proved of any use. Iodides and thyroid have been tried without effect. It is well to encourage the child to use his muscles freely, short of fatigue.

Two other even rarer forms of chronic progressive generalised inflammation of muscles have been described by F. E. Batten¹ as myositis fibrosa and dermato-myositis.

In **Myositis Fibrosa** the muscles undergo a fibrous change, without bony transformation. The disease, which begins in infancy, is characterised by great debility and by a generalised flexion with rigidity of the neck, back, and limbs. It is usually slowly progressive, but some cases of gradual recovery have been recorded.

Dermato-Myositis is a chronic inflammatory condition of the skin, subcutaneous tissue, and muscles which generally begins acutely with redness and swelling of the superficial parts and tenderness of the muscles. As the cutaneous swelling subsides, the muscles are left hard and infiltrated for some time. Successive attacks occur, and leave the skin thickened and inelastic, and the muscles fibrous and hard. Complete recovery may take place after months of illness, or the disease may progress to a fatal issue. The treatment consists in rest in bed and the administration of salicylates and antipyrine. Hot baths, massage, passive movements, and suitable splints are also useful in the later stages of the disease.

Myasthenia Gravis.

Myasthenia gravis is also a very rare disease in childhood. The following is a description of the only case which one of us (J. T.) has seen. She was eleven and a half years old and was referred by Dr Lumsden of Denny. "The child showed in a most striking way the readily produced and exaggerated muscular fatigue which is characteristic of this disease. She complained of extreme tiredness on walking, and of inability to go upstairs. This had begun four months before I saw her, and was at first attributed to stiffness from rheumatism, though there was no pain. Soon after, it was noticed that towards afternoon her face lost all expression and the eyelids drooped. During sleep the mouth opened owing to falling down of the

¹ *Clin. Soc. Trans.*, 1904, xxxvii., 12 ; and *Proc. Roy. Soc. Med.* (Neurol. Sect.), 1912, v., 103.

lower lip. The neck muscles were early affected, and soon became so weak that she had to give up doing anything that required her head being held forward. Lordosis also developed, and the weakness gradually spread to the other muscles of the body, though they showed no apparent wasting. When walking she often fell down 'because she felt quite done.' Her hands remained fairly strong, though she could only raise them above her head with difficulty by swinging them up, and could not keep them so for more than a few seconds. Similarly, she could not place her foot on a chair except by swinging the limb up from the hip, and when it rested on the chair she could not move it at all. When laid on the floor, the child had great difficulty in rising, and her attempts to do so resembled those of a patient with pseudo-hypertrophic paralysis. The speech was scanty, indistinct, and lisping, but not nasal, and speaking seemed to tire her. Three weeks before her death, which took place five and a half months after I saw her, the muscles of mastication became so quickly tired that she had to stop her meals long before she was satisfied; and, during the last week of life, respiration was distressingly difficult. Sensation remained intact, the knee-jerks and other reflexes were normal; the muscular sense and power of co-ordination were also unaffected and there were no fibrillary twitchings.

"One of the most striking features of the disease was the great variation at different times in the degree of debility. Until near the end, the child could get out of bed easily in the morning without assistance, hold herself erect, and walk fairly well; her face also looked fairly normal. Later in the day the eyelids drooped, the face lost all expression, and she walked and did everything else with increasing difficulty. Before evening she had to be lifted into bed."

The thymus is said to be enlarged in 90 per cent. of the cases.

No *treatment* has any effect on the symptoms, but it is said that some cases have recovered.

Muscular Dystrophy (*The Myopathies*).

The term "muscular dystrophy" is used to include a number of clinical types which have in common a progressive weakness, associated with changes in the affected muscles

without any lesion of the spinal cord. The paralysis is unaccompanied by pain or any other sensory abnormality; and the affected muscles, unlike those in some spinal paralysis, show neither fibrillary twitchings nor the reaction of degeneration. Their reaction to both galvanism and faradism is simply diminished. The knee-jerks and other deep reflexes are abolished when the muscles concerned are affected by the disease. The mental condition is either normal or slightly impaired. The changes in the affected muscles consist in atrophy and pseudo-hypertrophy, and in a few instances there is some temporary true hypertrophy.

The causes of muscular dystrophy are entirely unknown, but the disease is frequently seen in more than one member of a family, and the tendency may be transmitted to the next generation by the unaffected female members.

Some of the clinical types are fairly well defined and present curious and interesting differences, but transitional cases are not uncommon. Which of these clinical varieties are merely different phases of the same morbid condition, and which, if any, represent a different disease, is a difficult question which need not be considered here.

Pseudo-hypertrophic Paralysis.—Much the commonest and most striking of the myopathies met with in childhood is the pseudo-hypertrophic type. The patient who is practically always a boy, is born in apparently perfect health, and only shows definite evidence of the disease when he is about five years old or within a year or two of that age. In some instances he has seemed quite well and strong up till this time, but in others a history is given of his having been unaccountably late in walking, or of having always had difficulty in mounting stairs. As the disease progresses, the child's parents notice that he is getting weaker on his legs and is tired after the slightest exertion; also that he is easily knocked over and slow in rising. On examination, some of the muscles, especially those of the calf, the glutei, and the infraspinati, are found to be abnormally large (Fig. 263); others such as the thigh muscles, the lower part of the pectoralis major, the biceps, and the latissimus dorsi, are diminished in size; while others again, like those of the face, neck, forearms, and hands are unaffected. In advanced cases, there is usually some pointing of the feet. The child's attitude in standing is peculiar. He holds his

head up and his shoulders squared, while his back shows a marked lordosis. He plants his feet widely apart and has a waddling gait.

The way the child raises himself from the ground is particularly characteristic (Fig. 264). He begins by straightening himself out and rolling round on his face; then he slowly

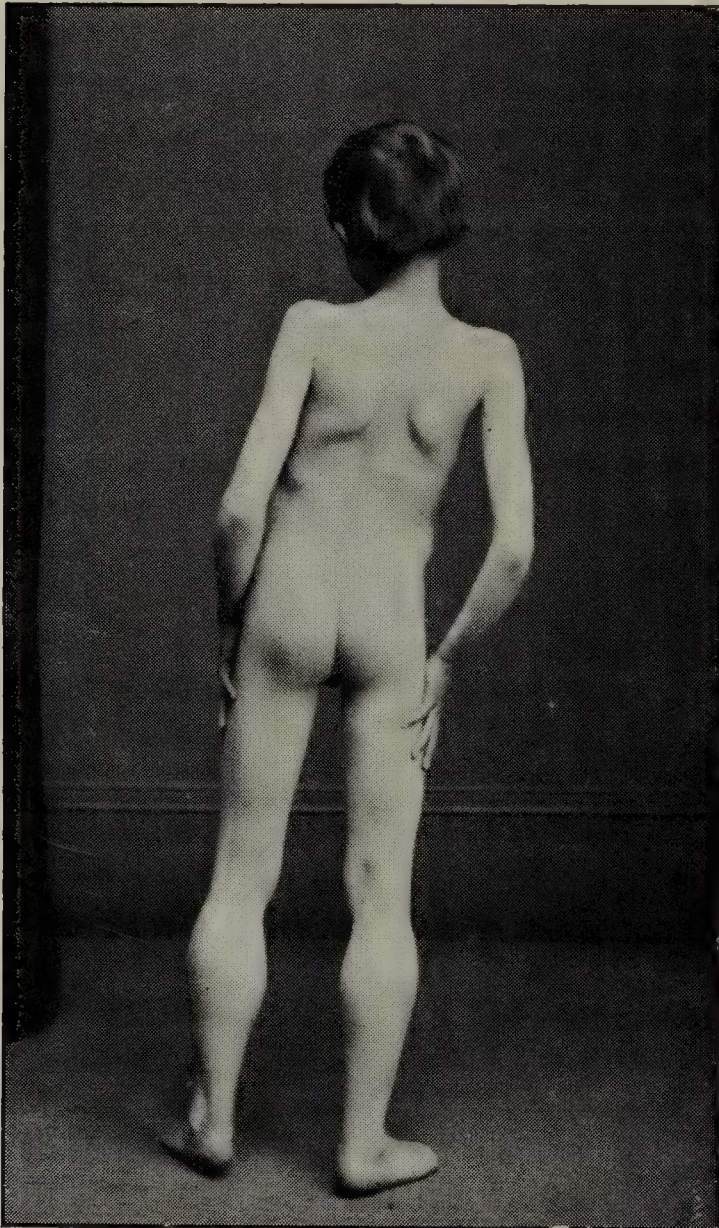


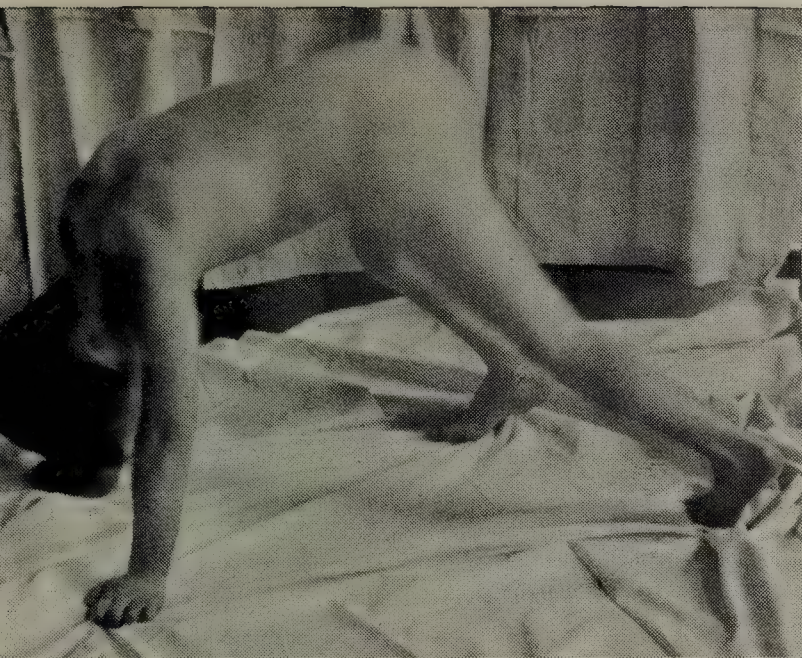
FIG. 263.—Pseudo-hypertrophic Paralysis.

flexes the thighs till he gets on to all-fours, and laboriously struggles into the erect posture. In doing so, after getting on to his feet, he transfers his hands to the front of his thighs and raises his shoulders gradually into the erect position by "climbing up the thighs." Finally the head and shoulders are jerked backward, so as to bring them behind the centre of gravity of the body, thus restoring the lordosis which is necessary for its balance. This way of rising from the ground, though characteristic of this disease, is not altogether peculiar to it. It is seen occasionally, though to a less degree, in various other forms of weakness of the lower limbs.

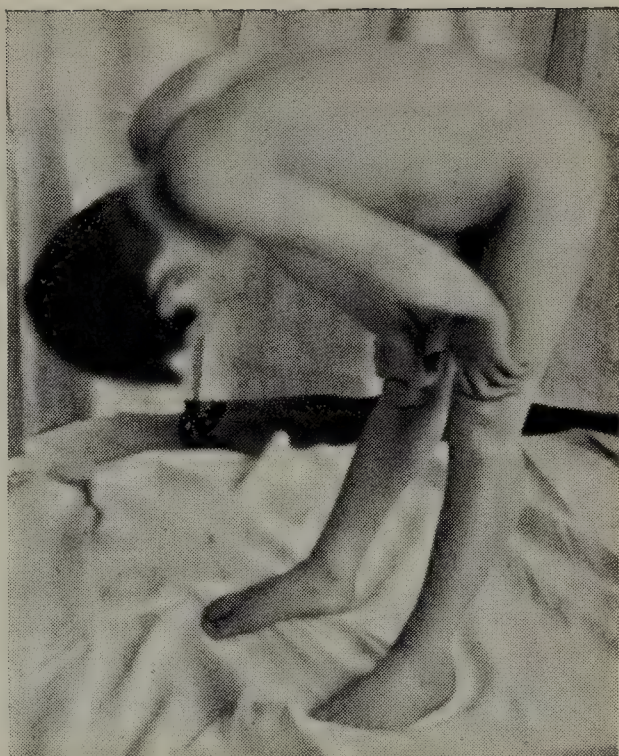
The extreme weakness of the muscles of the shoulder-girdle gives rise to another striking symptom. This is demonstrated by trying to lift the child by means of a hand placed in each axilla. When this is done, the limp axillary muscles fail entirely to support the weight of the body, which slips down through the hands, while the shoulders are carried up till they are on a level with the ears.

As years pass, the muscular weakness increases in degree and extent; but often its increase is intermittent, and it may

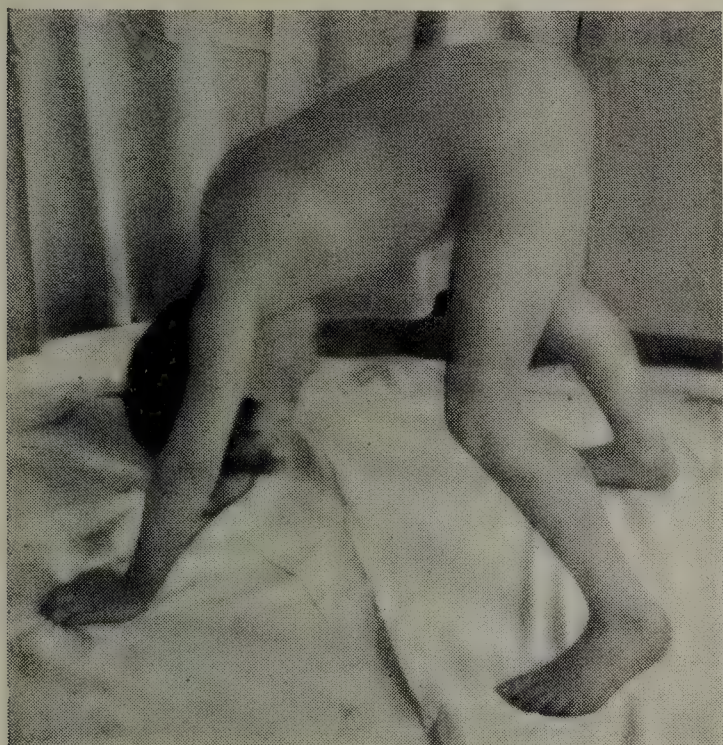
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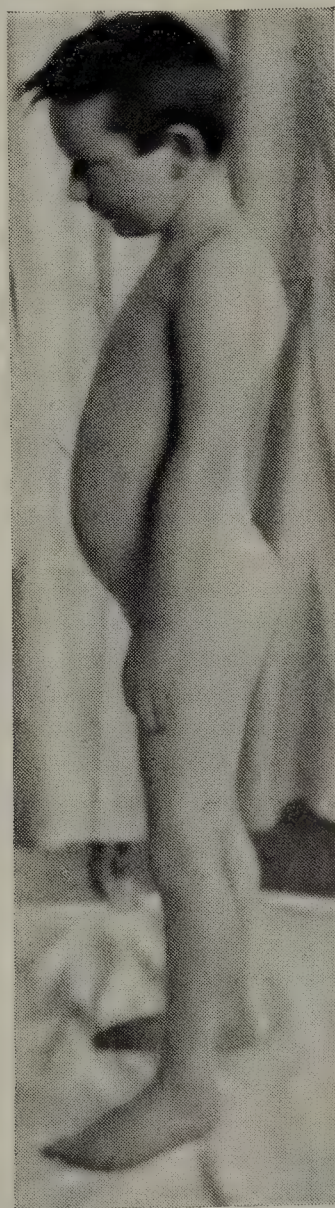
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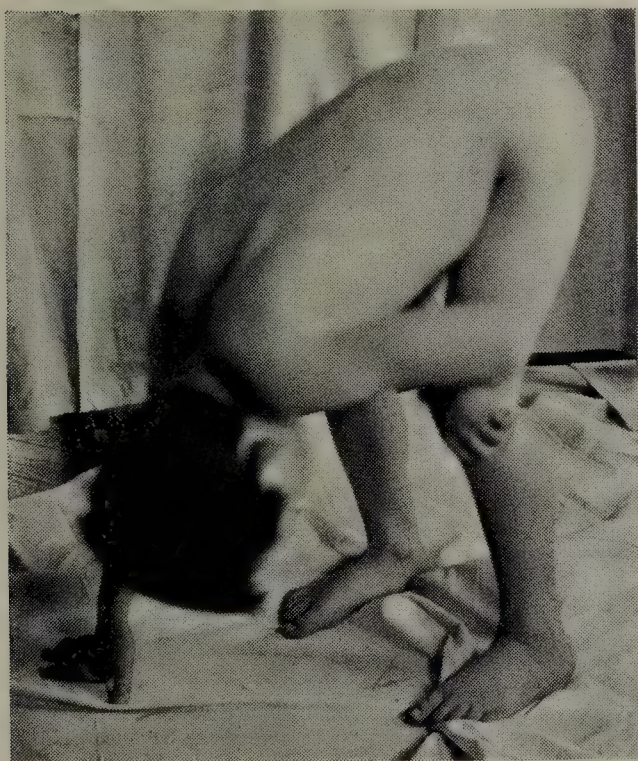


FIG. 264.—Showing method of rising from ground and lordosis in the case of a child suffering from Pseudo-hypertrophic Muscular Dystrophy.

be arrested sometimes for months or even years. By the time the patient is fifteen or seventeen years old he is in a state of profound weakness, and because of general rigidity of the muscles on account of their replacement by fibrous tissue, he is often completely bed-ridden. Death usually occurs rapidly at the end from some respiratory complication.

Treatment.—Although no successful treatment of the disease has been discovered, the progress of the paralysis can be delayed by massage, and especially by encouraging the moderate but persistent use of all the muscles, and by not allowing the patient to be too long in bed. The general health must also be maintained by attention to the diet and digestion. Occasionally tenotomy of the tendo Achillis is necessary to make walking easier.

The **Facio-Scapulo-Humeral Type** (Landouzy - Dejerine) is characterised by weakness of the orbiculares oris and palpebrarum. This may be present at birth, so that the baby cannot suck properly and is unable to close his eyes in sleep, and it usually remains stationary, or nearly so, for life. There is also weakness and wasting of the muscles of the shoulder-girdle, pelvis, thighs, and upper arms. These may become affected during childhood, but often are not so until well on into adult life. The disease affects both sexes equally, and is hereditary and familial.

The **Juvenile or Scapulo - Humeral Type** is much the rarest form of myopathy in childhood. Apart from the face not being affected, it resembles the Landouzy-Dejerine type in its distribution and in most of its other characters. The affection of the shoulder-girdle, which is its earliest manifestation, may begin as early as the sixth or seventh year. The progress of the disease is very slow and it does not endanger life.

Myotonia Congenita (*Thomsen's Disease*)^{1, 2}

When myotonia congenita begins in early childhood it leads to a striking over-development of all the muscles. The hypertrophy may be so noticeable that the case looks like one of pseudo-hypertrophic paralysis. On closer examination, however, this mistake is easily avoided, for the muscles of the arms and

¹ T. W. Griffith, *Quart. Journ. Med.*, 1911-12, v., 229.

² L. Findlay, *ibid.*, 493.

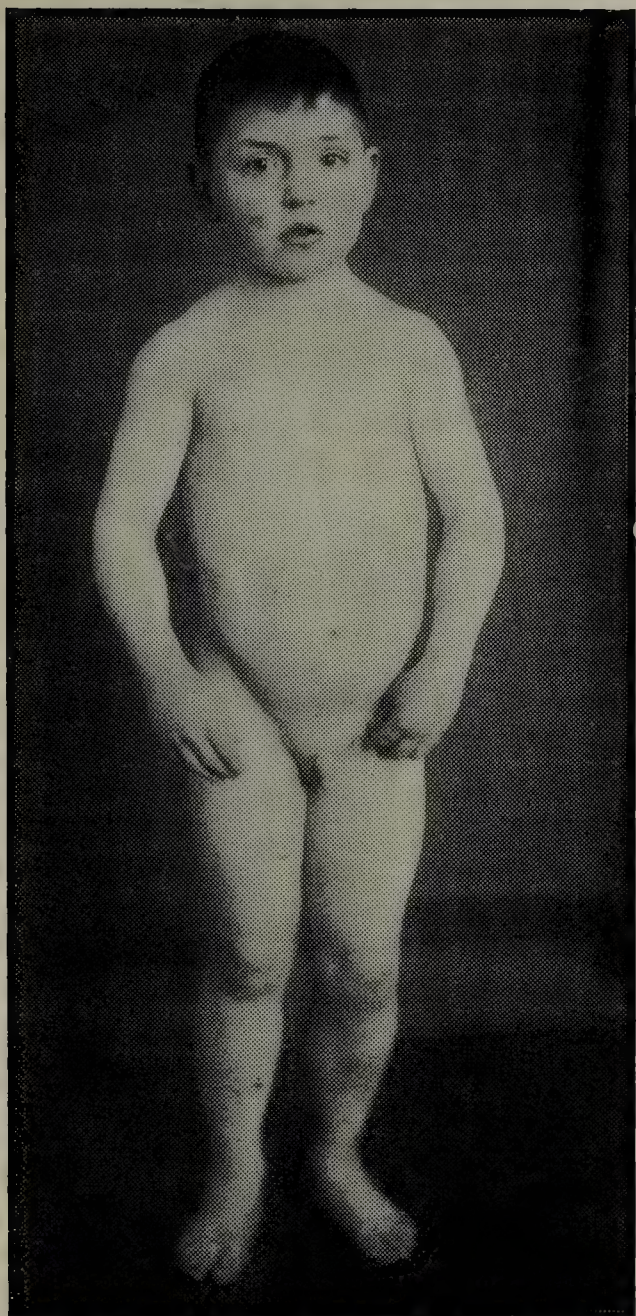


FIG. 265.—Myotonia Congenita. (Boy of 6 years.)

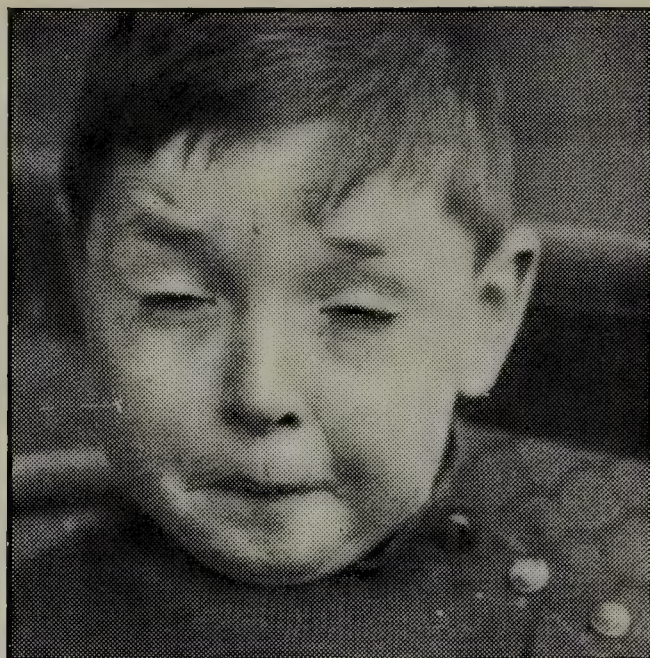


FIG. 267.—Trying to open eyes.

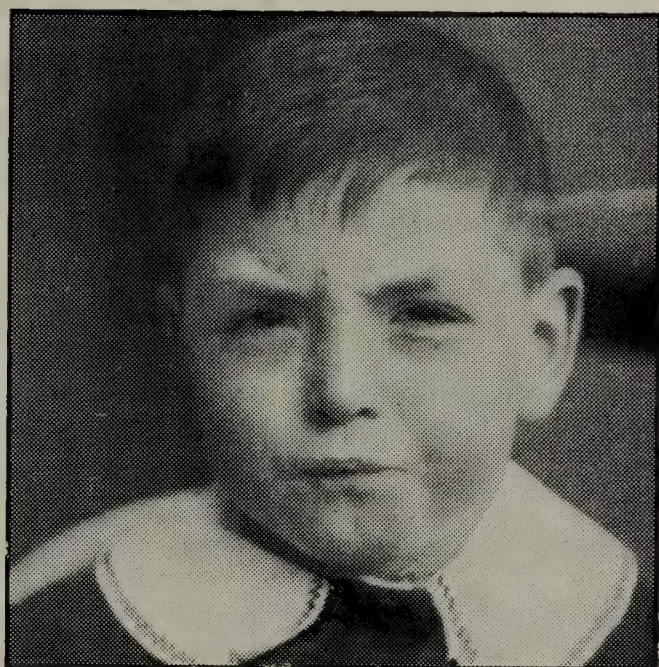


FIG. 268.—Eyes beginning to open.



FIG. 266.—Eyes closed.



FIG. 269.—Eyes open at last.

hands and those of the neck and trunk are quite as much enlarged as those of the legs (Fig. 265); and their strength is increased as well as their size. Other signs of pseudo-hypertrophic paralysis, such as loss of the knee-jerks, are also lacking.

The first thing that the mother notices will probably be that, after closing his eyes tightly during a fit of sneezing, the baby takes an extraordinary time and much effort in getting them open again. This symptom is easily demonstrated in an older child if you tell him to shut his eyes firmly and then to open them. To do so takes him about half a minute and much grimacing (Figs. 266 to 269).¹ When he repeats the process, however, it becomes easier each time; and after doing it four or five times in succession he can open his eyes quite normally.

As the child grows older, though he walks steadily and uses his hands as well as other children, if he is allowed to take his own time, his limbs are curiously useless when he tries to do anything in a hurry. He cannot start running quickly like other children; but, though his limbs seem very stiff and helpless when he begins to move, he gains control over them after a few steps, and can then run as well as other boys. A similar difficulty in beginning to move is seen in grasping and in all other voluntary movements, and it passes off quickly when the actions are repeated several times. All the other functions of the body are normal and the intellect is unaffected.

The disease, which is a very rare one, is not affected by any form of treatment; but, as the patient grows older, he learns in time how to minimise the inconvenience of his infirmity and to prepare for expected exertions by preliminary movements.

Sterno-mastoid Tumour (*Hæmatoma of the Sterno-mastoid*).

In the out-patient departments of Children's Hospitals we often see babies who are brought on account of a painless movable lump in the substance of one or other sterno-mastoid. The swelling varies in size, being in some cases like a hazel-nut and in others much larger. It is generally situated about the middle of the muscle, and is much commoner on the right than on the left side. The mother has usually discovered it by

¹ J. Thomson, *Edin. Med. Journ.*, Mar. 1916, 216.

chance while washing the baby, but sometimes her attention has been drawn to it because she has noticed that the head was being held inclined to the affected side. The tumour may be found as early as the first week, but often it does not attract attention until the child is from four to six months old or later. When discovered early, there is sometimes a definite hæmatoma present and the swelling is rather soft and fluctuates; later, however, it has a hard fibrous consistence. Whether there has been a hæmatoma at first, or whether, as more frequently happens, there has been no sign of this, extensive chronic myositis always develops and often goes on until a hard lump of fibrous tissue is formed—a sort of “muscle-callus.” The localisation of the fibrous swelling into an oval mass, and the comparatively slight separation of the torn ends of the muscle, are due to the complete sheathing of the sterno-mastoid by its fascia. The swelling is not necessarily permanent. We ourselves have observed it gradually disappear, and the fact that it is so seldom met with in older children is further evidence that the new-formed fibrous tissue may be absorbed.

It is generally accepted that the sterno-mastoid tumour arises from tearing of the muscle during delivery. As long ago as 1892 Herbert Spencer,¹ from histological examination, demonstrated hæmorrhage and rupture of muscle fibres in infants dying soon after birth. There is no doubt, too, that there has been a difficult birth in the majority of the cases. In many instances the presentation has been a breech or arm, and other evidence of injury, *e.g.*, Erb’s paralysis or cephal-hæmatoma, has been present. Nevertheless, on rare occasions no history of a difficult labour is obtained, and Rettig² has suggested that it may arise during intra-uterine life, but from what cause he does not say. Within recent years it has been considered analogous to Volkmann’s ischæmic paralysis. Middleton,³ who supports this view, holds that it is due to stoppage of venous and not arterial circulation and is thus of the nature of a venous infarction with necrosis of muscle and fibrous replacement. This author believes that he has experimental proof of his view, but the fact that there is in the early stages definite swelling, and that as a rule this swelling

¹ Herbert Spencer, *Journ. Path. and Bact.*, 1892, i., 113.

² Rettig, “Dissert. Halle,” quoted *Arch. Kinderheil.*, 1911, lv., 285.

³ D. S. Middleton, *Brit. Journ. Surg.*, 1930, xviii., 188.

disappears, seems to us to remove the sterno-mastoid tumour from this category of lesion.

Beyond simple measures for keeping the head still in the early stages and not allowing it to be bent over to the affected side, no special treatment is called for. Strong massage probably increases the fibrous tissue formation, but gentle rubbing may possibly be of service. In time the tumour lessens, and it disappears entirely within a few months or at most within the first year of life; in a majority of the cases no bad effects are left.

The chief importance of the lesion depends on the fact that in more than a third of the cases, and probably in most of those in which the muscle has been extensively torn, shortening results and a condition of wryneck develops which necessitates subsequent operation. In most, if not in all, of the cases there is also some arrest of development of the face on the affected side. This may not appear distinctly for a year or two, or it may be quite obvious within the first twelve months.

Whenever it is found that the muscle is becoming shortened, the advisability of a surgical operation should be considered. This consists in cutting down on the muscle, preferably near its sternal end, and dividing freely not only the damaged sterno-mastoid but also any other structures which are interfering with the head assuming its normal position. There is no great hurry for this to be done, but it is probably advisable that it should be undertaken within a few months of definite contraction being noticed. Unless the lesion is unusually extensive and severe, the result is generally quite satisfactory.

Ischæmic Myositis (*Volkmann's Paralysis*).

When a limb has undergone prolonged excessive pressure from the unskilful application of a bandage during the treatment of a fracture or other injury, permanent paralysis and contraction of the muscles occasionally results. This, as Volkmann pointed out, is due to coagulation-necrosis of the muscular tissue from interference with the arterial blood supply.¹

¹ W. Harris, *Brit. Med. Journ.*, 26th Sept. 1908, ii., 919; D. M. Greig, *Edin. Med. Journ.*, June 1910, N.S., iv., 498; Purves Stewart, *Brit. Med. Journ.*, 17th Aug. 1918, ii., 151.

Although the ischæmic myositis is the main and sometimes the only lesion present in these cases, there is occasionally an ischæmic neuritis also, involving the chief nerves of the affected limb. When this takes place severe pain, hypersensitiveness, or loss of sensation occurs, and the skin may become glossy or even ulcerate at the site of the pressure or on the hand or foot.

In the majority of cases Volkmann's paralysis is found in the upper limb, and has occurred during the treatment of a

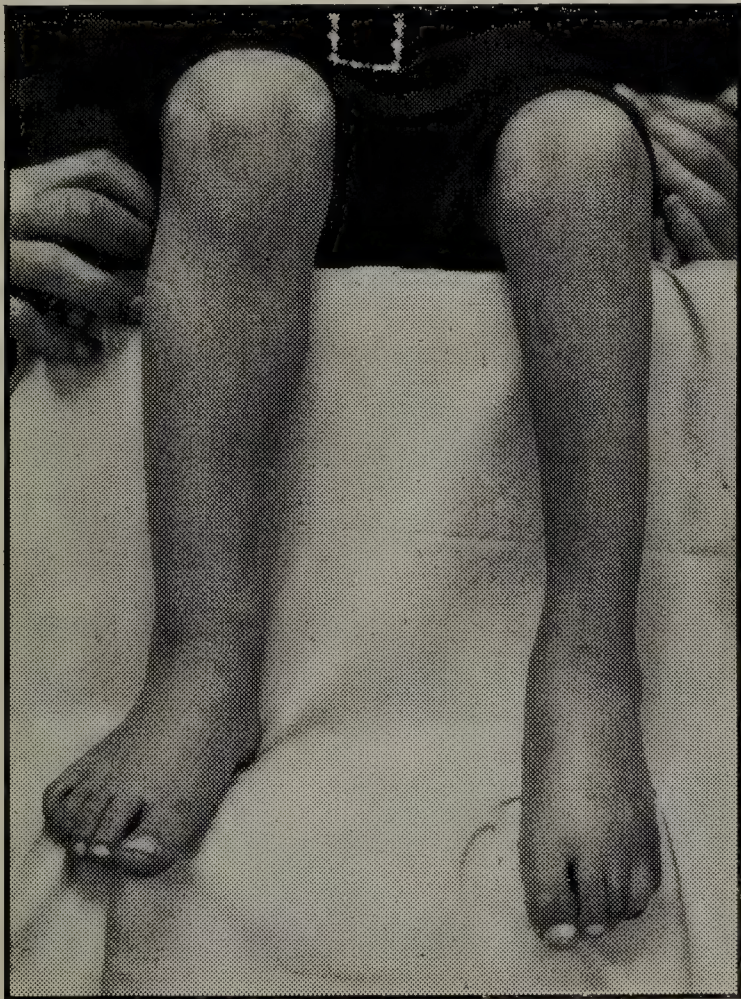


FIG. 270.—Ischæmic Myositis, after fracture of femur.
(Boy of 9 years.) (Mr D. M. Greig's case.)

fracture or other injury in the region of the elbow; but a similar condition is met with, though much less frequently, in the leg during the treatment of fractures of the femur (Fig. 270).

Symptoms.—Soon after the too tight bandage is applied, the hand (or foot) becomes swollen and livid and there may be severe pain. If the constriction is removed within a day or two the circulation rapidly recovers; but, even by this time, permanent damage may have been done. At first the muscles seem only paralysed, but very soon rigid contracture begins and it increases for several weeks. The muscles become very hard

but do not waste much ; they give the reaction of degeneration, and the deep reflexes are lost.

When the upper limb is affected the wrist becomes firmly flexed and the fingers are bent into the palm. The thumb often escapes, because it has a long flexor of its own. In the lower limb the toes are flexed on the sole and the foot is pointed.

Prognosis.—Any sensory symptoms present may recover in time, but the injury to the muscles is beyond repair.

Treatment.—Some slight improvement may be hoped for from prolonged massage, hot baths, passive movements, and the use of suitable splints ; and when the lower limb is affected tenotomy is sometimes of service.

CHAPTER XXXIV

DEFECTS AND DEFORMITIES OF THE BONES

Achondroplasia (*Chondrodystrophia Fœtalis*).

Pathology and Etiology.—Achondroplasia has been described as a foetal disease, in which there is “an absence, arrest, or perversion of the normal process of endochondral ossification, of the most definite and universal character, in every element of the skeleton in which the process normally takes place in intra-uterine life.”¹ Its effects are limited to those areas where bone is replacing cartilage—the normal pre-ossification process being inhibited by an unknown influence. According to A. Keith, this may be of the nature of a hormone.² The period during which it acts is believed to be between the third and sixth month of foetal life. At birth, accordingly, we find mainly the results of a past disturbance, and not an advancing process as in congenital syphilis and cretinism. Occasionally the disease occurs in several members of a family, or in a father or mother and child, and some instances have been observed in which cretinism, infantilism, or other forms of dwarfing have been found in the same family.

Clinical Features.—There are various degrees of severity of the disease. If the child is profoundly affected, hydramnios is often present, and the labour is generally premature. In such cases, if the baby is born alive, he is often so weakly that he dies within a few days. Not uncommonly hydrocephalus is present at birth or develops later. We have met with no other congenital defect in these cases except in one in which there was congenital pyloric stenosis. From this the child recovered, but he died later from chronic hydrocephalus. When the achondroplastic child survives infancy, the subsequent development of the muscular, cutaneous, and reproductive systems is quite

¹ Symington and Alexis Thomson, *Lab. Rep. Roy. Coll. Phys. Edin.*, 1892, iv., 238.

² Arthur Keith, *Journ. Anat. and Physiol.*, 1913, xlvii., 189.

normal, and the intellect is unaffected. The chances of a long life are probably as good in these children as those in an ordinary individual—except in the case of women who become pregnant.

The most striking feature in achondroplasia is the disproportion between the size of the trunk and that of the limbs (Figs. 271, 272, 273, and 274). The trunk is of fairly normal length, but rather narrow from the shortness of the ribs and the contraction of the pelvis.



FIG. 271.—Achondroplasia.
(Still-born infant.) (Dr H.
Rainy's case.)



FIG. 272.—Achondroplasia.
(Girl of 4 years.)

The arms and legs are much too short, especially in their proximal segments—being often in adults little more than half the normal length. The long bones are thick as well as short, with very broad epiphyses; they are also curved, the curves representing merely exaggerations of those normally present, and knock-knee is common. On X-ray examination the ossification of the bones (*e.g.*, of the carpus) in childhood is found to be very much retarded (Fig. 273). The limbs are often

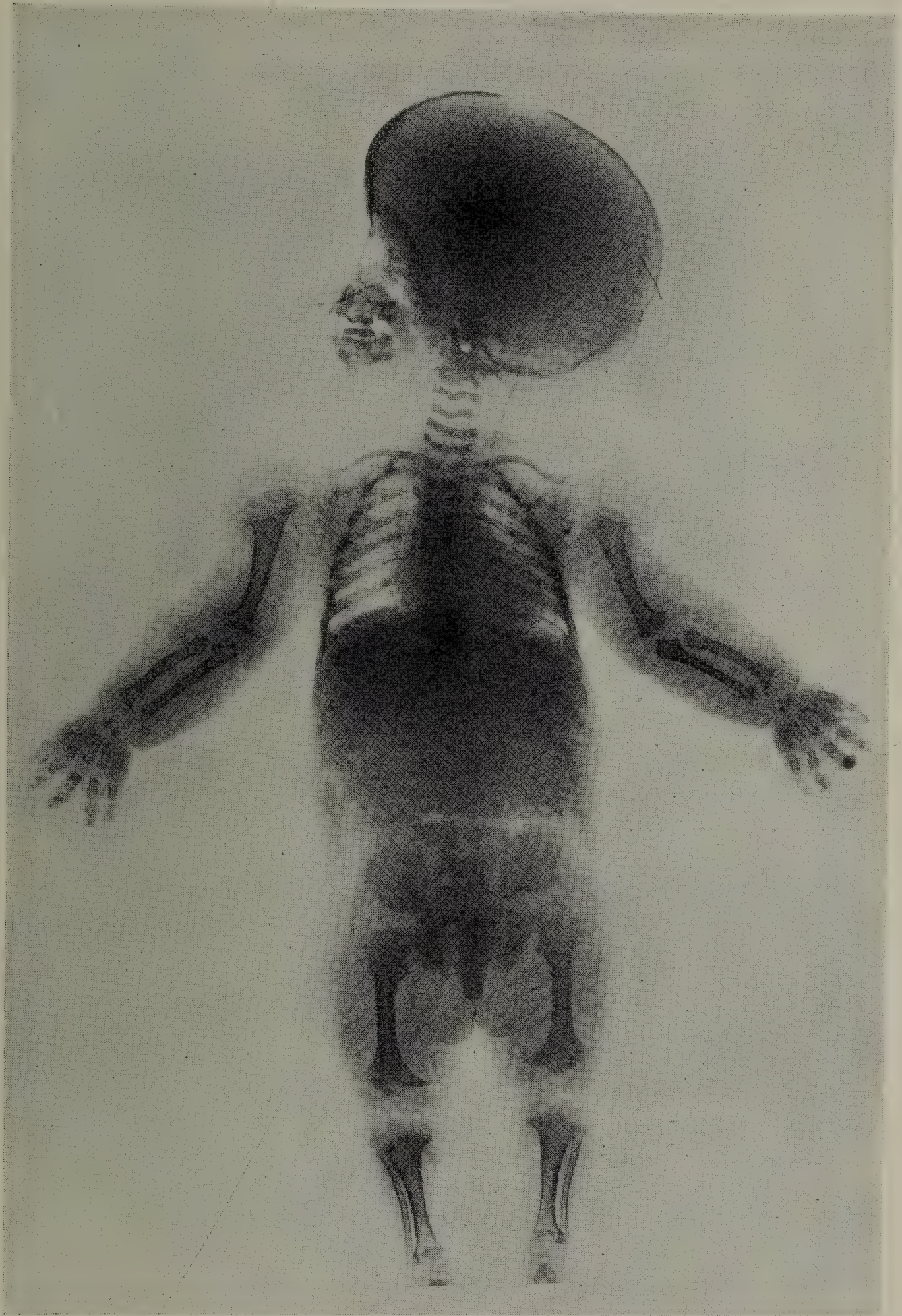


FIG. 273.—Skiagram of Skeleton of Achondroplastic Male Child
aged 2 years and 7 months.

surrounded by deep sulci, as if the skin and soft tissues were on an ampler scale than the length of the bones required. In Fig. 274 is shown one of the statuettes of the Egyptian god Ptah which are frequently found in mummy cases. It shows



FIG. 274.—Statuette of the Egyptian god Ptah-Sokar.

the clinical characteristics of achondroplasia in a striking manner.

The hands are always peculiar.¹ They are relatively broad and very short (Fig. 275). The fingers are thick and short, and the individual digits often look all about the same length. The



FIG. 275.—Achondroplasia.
(Hand of boy of 5 months.)

most striking peculiarity, however, is the want of parallelism in the fingers. When the hand is laid flat, so that the palm is not hollowed, they spread out in such a way that their ends are separate from one another, and not close together as in the normal hand. The index and middle fingers usually curve to the radial, the ring and little fingers to the ulnar side. For this condition the term “trident hand” has been proposed.²

The shortness of the lower limbs gives the child a peculiar waddling gait; and soon after beginning to walk he develops a deep lordosis, which increases as he grows older (Fig. 272).

¹ J. Thomson, *Edin. Med. Journ.*, June 1893, xxxviii., 1111.

² Pierre Marie, *Presse Médicale*, 14 Juillet 1900, 21.

The head is rather large, the cranium being high and bulging in front and at the sides; and the root of the nose is distinctly depressed. The increased height and breadth are due to an overgrowth of the membranous elements of its roof and sides to compensate for the diminished size of the cartilage-formed bones of the base. In the full-time achondroplastic infant the basi-occipital and the basi- and pre-sphenoid are fused into one mass, and the ossification in the pre-sphenoid is arrested. The suture between the basi-occipital and the basi-sphenoid is closed, and those between the ex-occipital and the other components of the occipital bone are almost so; the foramen magnum is little more than one-half the normal size. The extreme shortness of the base which these changes produce gives rise to a diminution of the naso-pharyngeal space. They also contribute to the nasal retraction, which is also rendered more noticeable by the bulging of the forehead (Fig. 273). At birth the tongue often protrudes from the mouth, but this does not occur in later life.

Diagnosis.—*At birth* the deformity described above forms a picture which is easily recognised. The appearance of these infants has often been described as “cretinoid,” and aptly enough, because they are certainly like cretins in several particulars. The likeness, however, is to adult cretins, and not to cretin babies. In infancy, even severe cases of sporadic cretinism do not present much, if any, disproportion of the limbs and trunk, nor the typical physiognomy of the disease, except to a very slight degree (p. 504). The thyroid of the achondroplastic child is distinctly felt, and he has no supra-clavicular swellings. He generally cuts his teeth early, and is not late in beginning to walk; while rickety dwarfs have almost always been very backward in both these particulars.

The achondroplastic *adult* is readily distinguished from the rickety dwarf by the symmetrical shortness of all his limbs, the absence of rickety distortion in the spine, limbs, thorax, and head, and especially by the peculiar shape of the hands.

Older achondroplasics differ from cretins in the normal temperature and texture of the skin, their muscular and intellectual vigour, and the natural development of the organs of sex.

No treatment has any effect on the disease.

Other Forms of Dwarfing with Short Limbs.

There are two other varieties of dwarfing, with short limbs and peculiar hands, which are apt to be mistaken for achondroplasia. They are both uncommon, and the pathology of neither of them, so far as we know, has been investigated.

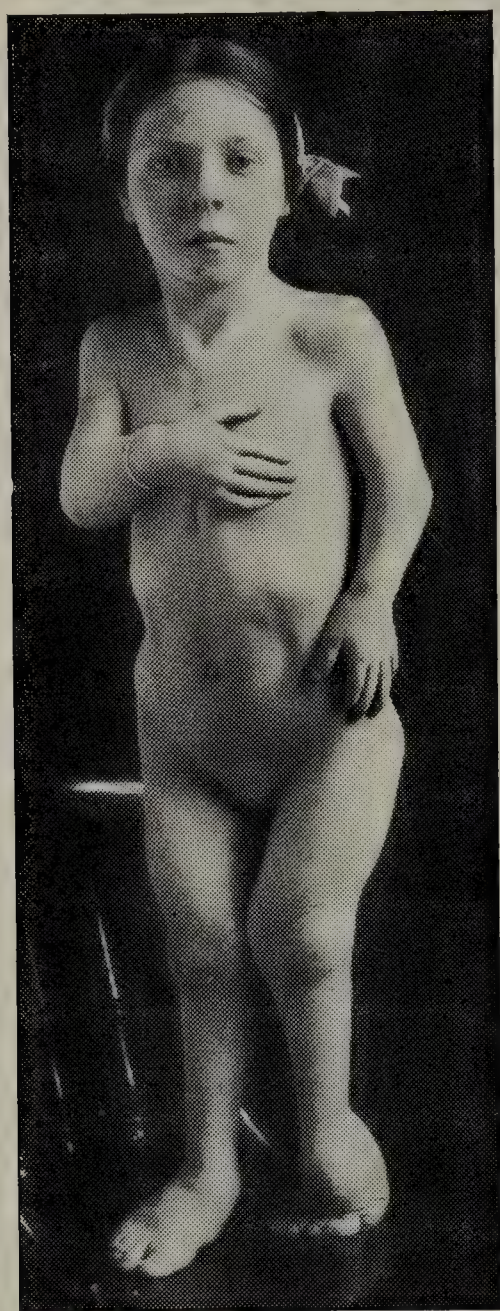


FIG. 276.—Short-limbed Dwarf.
(Girl of 9 years.)

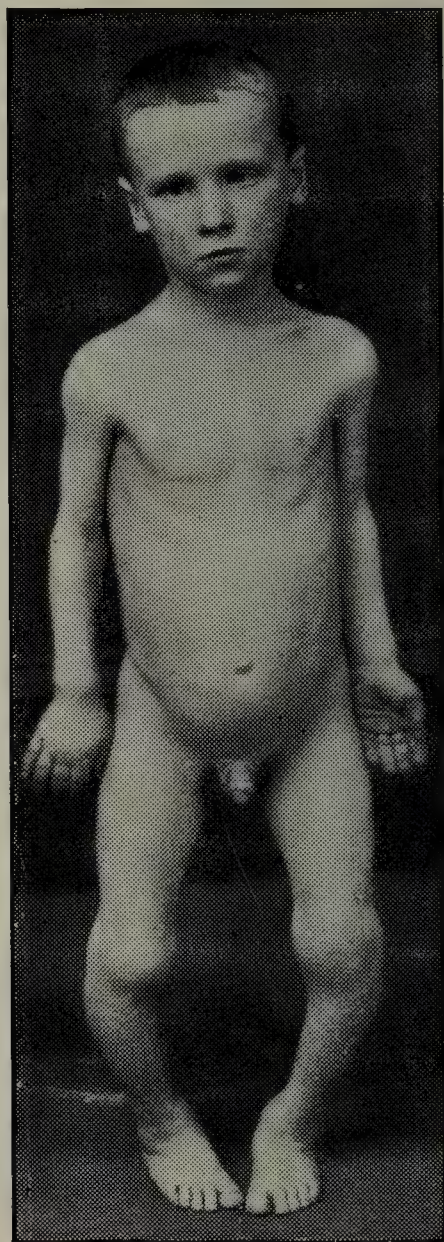


FIG. 277. — Short - limbed Dwarf. (Boy of 10 years.)

The most striking characteristics of the *first* of these (Fig. 276) may be described as follows:—

The deformity is present to its full extent at birth. The head is not obviously abnormal in its ossification, and has none of the usual achondroplastic characters. The auricles are sometimes peculiarly convoluted. The trunk and clavicles are normal in shape. The extremities, especially the upper arm

and thigh, are shortened, and there is usually knock-knee after the child has walked for some time.

The hands are peculiar and characteristic. The fingers do not straighten completely, and their extremities deviate towards the mesial line of the limb, instead of turning away from it as in achondroplasia. In order, therefore, to make them lie parallel and in close contact with one another, the surface of the palm has to be rendered convex. The ossification of the carpal bones is slightly in advance of the normal for the child's age. The feet are abnormally short, and there is a tendency to talipes



FIG. 278a.—Skiagram of Hand and Arm. (Same case as Fig. 277, at 12 years.)

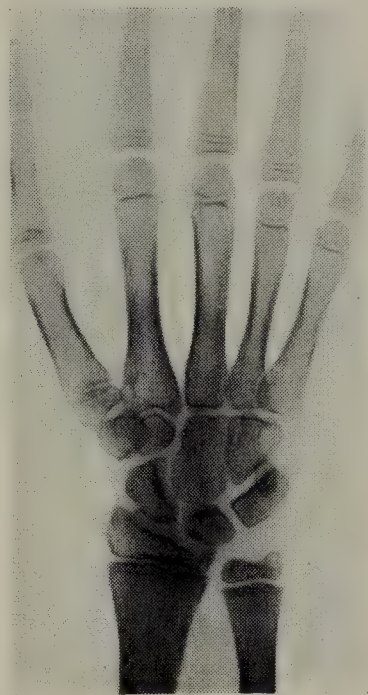


FIG. 278b.—Skiagram of normal Hand at age of 12 years.

equinovarus. The muscles are not abnormal, though poorly developed owing to the extent to which the abnormality of the feet has interfered with exercise. The intelligence is unaffected. In one instance the deformity occurred in twins; but no hereditary tendency to its occurrence has been observed.

With regard to the *second* type of dwarfing, several cases were described by Herringham and Drysdale in 1908¹ (Figs. 277 and 278a).

So far as can be gathered from these cases, and from four observed by one of us (J. T.), the main clinical features which characterise this disease, and distinguish it from achondroplasia and other forms of dwarfing, are as follows:—

(a) The deformities of the bones are not congenital in

¹ *Quart. Journ. Med.*, Jan. 1908, i., 193.

origin, the size and conformation of the limbs being normal at birth and for a few years after. The teeth come early and the fontanelle closes at about the usual time. During the latter part of the second, or in the third year of life, the child begins to suffer from rather severe pains in the lower limbs after walking, and very gradually the characteristic dwarfing of the hands and bowing of the legs develop.

(*b*) As the limbs continue to grow, the upper arms remain relatively shorter than the fore-arms. The deformity of the hands is, however, the most striking feature. Their growth becomes gradually arrested within a year or so of the onset of the pains in the knees and, at the same time, they grow in thickness. The whole hand becomes stunted and much thickened, and the fingers are thick, fleshy, and conical, but they show no deviation. On X-ray examination, great thickening of the phalanges is found, and their ossification, and that of the carpal bones, is very much delayed (Fig. 278*a*, p. 875).

(*c*) The lower limbs seem normal at birth, but from the time that the pains begin there is a tendency to bow-legs, and it gradually increases to a severe degree. The growth of the bones is also interfered with, and the limbs seem shorter than they are owing to the bowing.

(*d*) The head shows none of the characteristic peculiarities of achondroplasia, and the teeth are normal.

(*e*) The intellect and the sexual development are not interfered with.

(*f*) Thomson's three older cases suffered after the first year from well-marked rickety enlargements of the ends of the long bones and from beading of the ribs.

(*g*) Herringham and Drysdale describe three cases occurring in one family. Thomson's cases were father, son, and two daughters; no other relation was similarly affected.

Cleido-cranial Dysostosis.¹

The rare hereditary and familial abnormality known as cleido-cranial dysostosis, first described by Sir Thomas Barlow in 1883, affects the bones which are normally formed in

¹ T. Barlow, *Brit. Med. Journ.*, 15th Sept. 1883, ii., 509; Marie and Sainton, *Soc. Méd. des Hôp.*, 1897 and 1898; Schorstein, *Lancet*, 1899, i., 10; Geo. Carpenter, *ibid.*, 13; D. C. L. Fitzwilliams, *Lancet*, 1910, ii., 1464.

membrane, just as achondroplasia does those which originate in cartilage. It is much rarer than the latter condition.

Clinical Features.—Its most characteristic features are (*a*) an increase in the transverse diameter of the skull; (*b*) delayed ossification of the cranium; and (*c*) more or less extensive aplasia of the clavicles.

The skull during infancy is largely membranous, and its ossification is late in taking place. The anterior fontanelle may close in later childhood, or may remain widely open during adult life. As the cranium ossifies, bosses form on the frontal, parietal, and occipital regions. The head is large, brachycephalic, and flat posteriorly, and its base is shortened. The face is relatively small, the palate high and occasionally cleft, and the teeth irregular. The eyes are rather staring, from prominence of the supraorbital regions compared with the lower borders of the orbits, the nasal bones are short, and the lower jaw sometimes underhung.

The clavicles are defective. Sometimes one or other of them is altogether absent; generally, however, the sternal end is present, and often the central part only is wanting. Owing to the clavicular defect, the shoulders can be readily brought together so as to meet under the chin (Fig. 279).

The stature is rather short, and other defects of the osseous system, such as scoliosis, knock-knee, and club-foot, are sometimes present. Apart from the defect of those muscles which are connected with the defective clavicles, the muscular and other systems of the body are normal. The intellect is unaffected and the general health good. The condition is often hereditary.

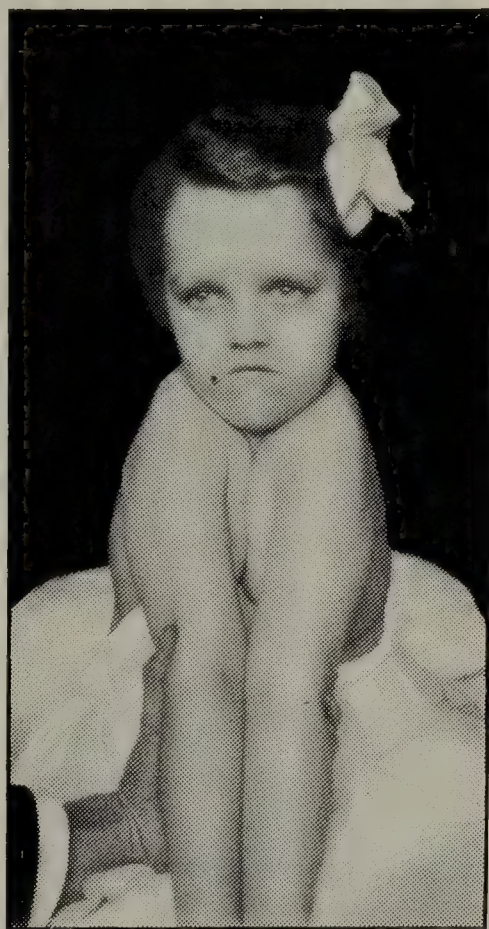


FIG. 279.—Cleido-cranial Dysostosis. (Sir Harold Stiles' case.)

Mollities Ossium.

Mollities ossium has been described as occurring rarely in childhood, though never apparently in early infancy. It affects both sexes, and may occur in more than one member in a family, but it is not hereditary.

The onset of the disease is gradual, and there is pain in the limbs in the early stages. The whole skeleton is involved, but the effects of the disease are chiefly seen in the lower limbs. These show bending of the bones, and occasionally fractures. There is great general debility, and the disease is difficult to distinguish from late rickets.

The patient has usually to be kept lying, and fresh air, sunshine, and the administration of cod-liver oil are indicated. Good results have been reported from the use of phosphorus and especially from adrenalin.

Fragilitas Ossium.

Brittleness of the bones, leading to their fracture from slight causes, is met with sometimes in infantile scurvy (Figs. 70 and 71, p. 233) and congenital syphilis, and is not uncommon in severe rickets (Figs. 85 and 86, p. 254 and Fig. 96, p. 281).

The term "fragilitas ossium" has usually, however, been employed in describing various rare congenital conditions, of which fragility of the bones forms a striking feature, but which have nothing to do with these other diseases. In a few of the cases the tendency to fracture only develops in later childhood; but in most of them it is present from infancy. Two groups of these cases seem to be fairly distinct clinical entities, and may be briefly described. These may be called "osteogenesis imperfecta" and "family fragilitas ossium with blue sclerotics."

Osteogenesis Imperfecta (*Osteopsathyrosis Infantilis*).

Osteogenesis imperfecta is the term most generally used at present to denote a rare intra-uterine disease, the subjects of which are usually still-born, although they occasionally survive and may live to adult life. Cases of the condition have often been described as intra-uterine or foetal rickets, and sometimes they have been published as achondroplasia. No cause of the disease has been discovered. One of us (J. T.) has known three cases to occur in one family.



FIG. 280.—Osteogenesis Imperfecta. (Girl of 2½ months.)

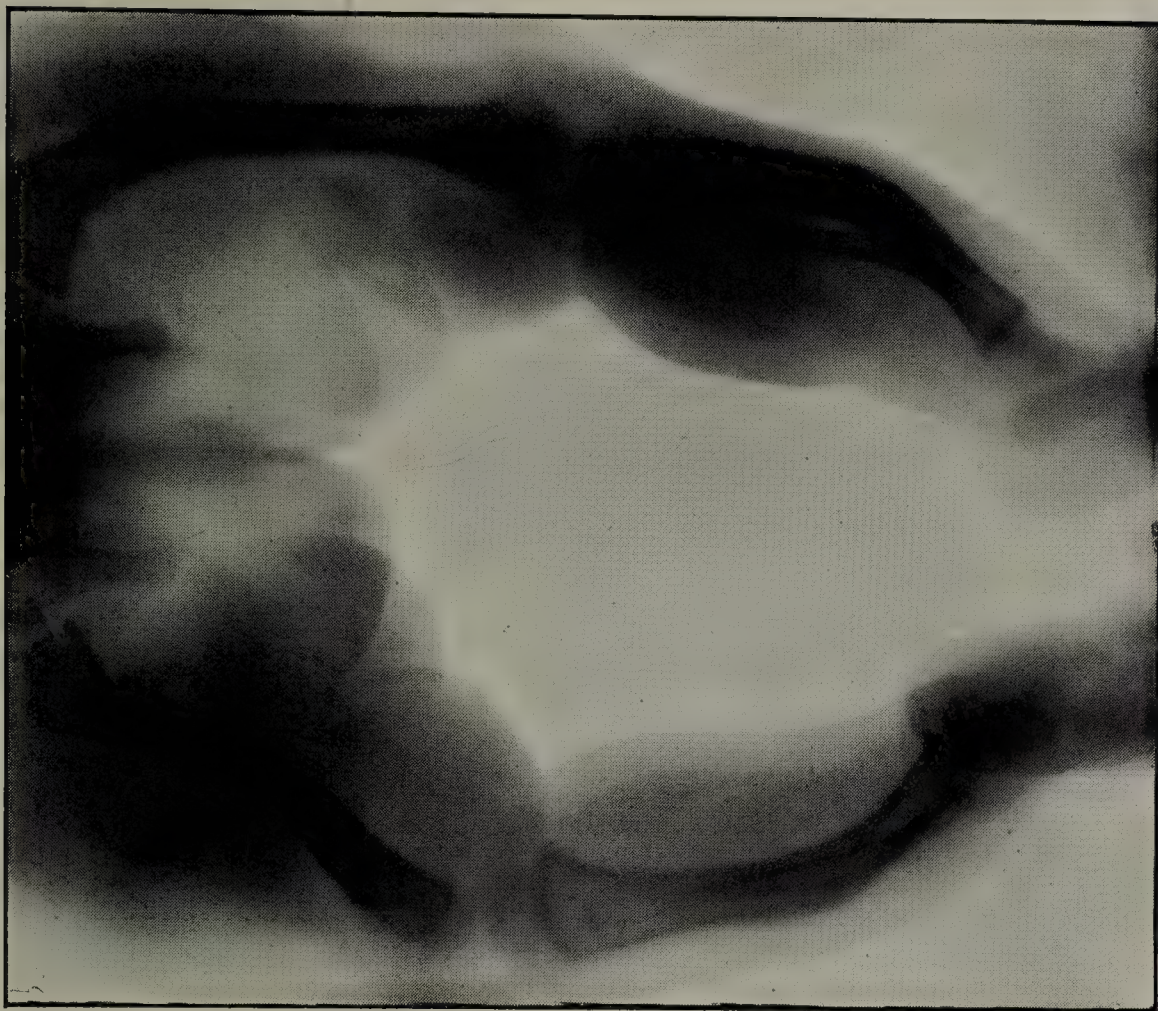


FIG. 281.—Skiagraph of lower limbs of the same child as in Fig. 280.

Pathology.—The bones are defective in calcification, and abnormally translucent on examination with the X-rays, and the periosteum is thickened. The disease depends on a congenital disturbance of the periosteal formation of bone. There is no enlargement of the epiphyses or beading of the ribs.

Symptoms.—When born, the children are fat and seem to be well nourished (Fig. 280). The cranial vault is very defective in its ossification, and shows large soft areas which remain membranous for many months. Generally there is, at birth, marked bending of many of the long bones (Fig. 281), often with fractures, of which there may be many. Some of these are evidently recent, and may have taken place during delivery, while others are of longer standing and are surrounded by callus. The trunk is dwarfed, but the limbs show little or no relative shortness, except that caused by the fractures and bending of the bones. In some cases there is an inability to straighten the elbows and to extend the fingers completely.

If the children survive, the fragility of the bones generally persists, and they are liable from time to time to fractures from slight causes. In a few undoubted cases there is only bending of the bones and no fractures, either before or after birth. The fractures usually recover without difficulty under ordinary treatment. In all the cases that we have watched for a number of years the growth has proceeded slowly, but the stature has remained far short of the normal. As the patient reaches later childhood there is a remarkable tendency for the twisted bones of the lower limbs to lose their curves and to become almost normal in shape.

The cranium is large for the size of the body, but in infancy its form is not specially characteristic. In many, if not in all cases, a peculiar type of cranial malformation develops. The skull is large and broad with an angular projection running round its posterior and lateral aspects just above the level of the auricles (Figs. 282 and 283). There is a small upper and a relatively long lower jaw. The nose and chin tend to be long and pointed. In the cases we have seen there has been no blueness of the sclerotics. The intellectual development of the children is normal or above the average. When they reach adult life they often look more than their age. No medical treatment has proved of any value.



FIG. 282.—Showing peculiar form of Cranium in Osteogenesis Imperfecta. Side view.
(Boy of $11\frac{1}{2}$ and girl of $8\frac{1}{2}$ years.)



FIG. 283.—Front view of same children.

Family Fragilitas Ossium with Blue Sclerotics.

During recent years a good deal has been written on a type of fragility of the bones which occurs in certain families, and is accompanied by a peculiar blueness of the sclerotics, laxity of ligaments, otosclerosis, and other lesions. A full description of these cases, with a complete bibliography of the condition up to 1916, will be found in the late Dr Edith Bronson's very thorough paper on the subject.¹

According to N. Voorhoeve,² the morbid phenomena present are due to a hereditary inferiority of the mesenchyme. This accounts for the involvement of the bones and ligaments, the sclerotics, and, to a less extent, of the circulatory system.

Clinical Features.—(a) *Brittle Bones.*—The children appear normal and well formed at birth. They usually show no sign of rickets during infancy, and fractures very rarely take place before the second or third year. In later childhood some of the bones are often broken by moderately severe injuries. After puberty the brittleness of the bones diminishes, and fractures seldom occur. It is uncommon in this type to meet with so many fractures as are common in osteogenesis imperfecta. The bones show none of the ordinary characters of rickets, but present a rarefied condition with a deficiency of lime salts.

(b) *Abnormal Laxity of the Ligaments.*—Some cases are described as "loose-jointed"; and recurrent dislocations of the ankle, fingers, and other joints are not uncommon.

(c) *Peculiar Form of the Head.*—There is usually a characteristic shape of the head, which is recognisable in early childhood (Fig. 284). It is rather large, with a full forehead and prominent occipital and parietal regions. The shape differs from that in rickets and hydrocephalus, and shows no distinct bossing. This form of head is only seen in those members of the family who have a tendency to fractures.

(d) *Stunted Growth.*—The unaffected members of the families are generally of normal stature, while those affected are several inches below the average.

(e) *Blue Sclerotics.*—The incidence of blue sclerotics along with brittleness of the bones was first pointed out in 1900 by Eddowes.³ Their dark grey-blue colour is usually quite distinct

¹ *Edin. Med. Journ.*, April 1917, N.S., xviii., 240.

² *Lancet*, 1918, ii., 741.

³ *Brit. Med. Journ.*, 1900, ii., 222.

at birth. A similar or brighter blue colour of the scleræ is also seen in non-familial types of fragilitas ossium, in congenital syphilis, and in some cases in which there seems no special tendency for fractures to occur.

The blue colour is apparently the result of undue transmission of the colour of the choroidal pigment through an abnormally translucent sclerotic. It has been suggested that this trans-



FIG. 284.—Family Fragilitas Ossium with Blue Sclerotics. (Boys of 6 and 3 years.) Showing form of head. (Dr Bronson's cases.)

parency may be due to too little lime in the sclerotic tissue. Those members of the affected families who have blue sclerotics seldom escape having fractures or dislocations sooner or later.

(f) *Otosclerosis*.—This disease, which is known to be frequently associated with a variety of morbid conditions in the osseous and circulatory systems, has been found to occur in a considerable proportion of the affected members of these families who have reached adult life.¹

¹ W. S. Bryant, *Monatschr. f. Ohrenheilk.*, 1913, 436 and 584 (see Abstract by Dr J. S. Fraser in *Journ. Laryngol. Rhinol. and Otol.*, 10th Oct. 1915, xxx., 389).

(g) *Circulatory and Blood Diseases*.—In a small proportion of the published cases, hæmophilia has been present, and cardiac malformation has also rarely been noted.

(h) *Calcium and Phosphorus Metabolism*.—Dr Olive Macrae investigated a case at R.H.S.C., Glasgow, and found a much diminished daily retention of both lime and phosphorus. The retentions were of CaO and P_2O_5 .017 and .02 gramme per kilo body weight per day respectively, instead of the normal .05 to .1 gramme of each per kilo per day.

Treatment.—No therapeutic measures seem to have any effect in preventing the tendency to fracture of the bones.

Cod-liver oil and phosphorus have been often given for long periods without any good result.



FIG. 285.—Oxycephaly. (Girl of 10 months, who had recurrent dislocation of right eye.) Syndactyly.

Oxycephaly¹ (*Acrocephaly—Steeple-head*).

Oxycephaly is a rare developmental abnormality of the osseous system, with a curious malformation of the cranium as its most striking feature. As the commonest subjective symptom is impairment of vision, the condition has been mainly investigated by ophthalmic surgeons, and it was first described

by von Graefe.² There is occasionally a hereditary history, and more than one case may occur in a family.

Clinical Features. — The characteristic appearances are readily recognisable at birth in the worst cases. In others they only develop slowly between the second and sixth years. The degree to which the shape of the head is altered varies greatly. In some instances the deformity is so slight that it is only diagnosed on careful inspection and X-ray examination (Figs. 286, 287 and 288), while in others it is very obvious (Fig. 285).

The head is abnormally high and comes to a sharp point at the vertex. The superciliary and orbital ridges and the frontal

¹ H. Morley Fletcher, *Quart. Med. Journ.*, 1910-11, iv., 385.

² *Arch. f. Ophthalm.*, 1866, xii., Abth. ii., 114.



FIG. 286.—Oxycephaly. (Girl of 13 years.) (a) Side face.

(b) Front face

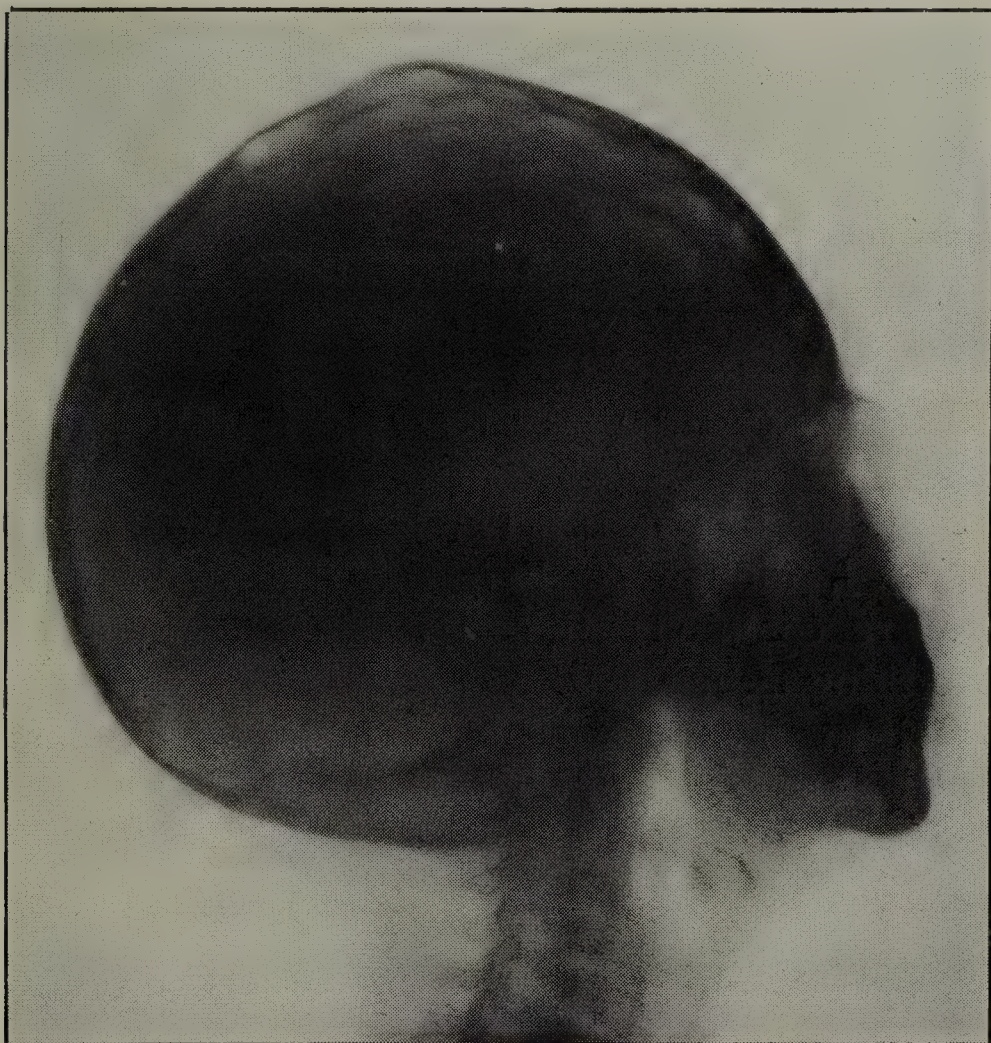


FIG. 287.—Skiagram of Cranium in Oxycephaly.

eminences are feebly marked. The eyes are protuberant and slanting—the inner canthus being higher than the outer. In some severe cases the exophthalmos is so great that spontaneous dislocation of the eye outside the lids occurs from time to time on slight exertion. Divergent strabismus is common, and rarely a convergent squint forms. Nystagmus is present in many cases. Vision is occasionally unaffected; but, in a majority of the cases, it becomes impaired sooner or later from the presence of optic atrophy. In the worst cases there



FIGS. 288*a* and 288*b*.—Oxycephaly. (Girl of 12 years.) (Dr R. Hutchison's case.)

may be complete blindness. The hearing is good, and taste is rarely affected, though smell is often entirely lost. Severe headaches are common; the palate is abnormally high, and mouth-breathing frequent.

Although the mental condition is unaffected, or even above the normal in some of the slighter cases, most of the severer type show some mental defect. This, however, is rarely severe in degree.

In many of the worst cases the hands are deformed, the fingers being webbed and badly developed. In some there is difficulty in full extension of the shoulder and elbow-joints.

On X-ray examination, the cranial bones show a pattern of rounded "digital" markings which look like impressions of the finger tips (Fig. 287). Premature synostosis of the sutures is found, especially of the coronal and sagittal. The orbits are shallow and are directed downwards and outwards. The sella turcica is wide and deep.

The **diagnosis** is generally easy from the peculiarity of the deformity; but cases are sometimes met with in which most of the characters of oxycephaly are present without pointing of the vertex.

Prognosis.—In the bad cases the children are generally feeble, but, in the slighter ones, the disease does not seem to shorten life.

The **cause** of oxycephaly is entirely unknown.

Treatment.—The headache is sometimes benefited by iodides. Trephining has given relief in some severe cases.

Hypertelorism.

The accompanying illustrations (Figs. 289 to 292) show the characteristic features of a rare congenital deformity of the head and face, which has recently been investigated and described for the first time by Mr D. M. Greig, who has given to it the name of "Hypertelorism."¹

Although severe examples of the deformity, like that seen in Figs. 289 to 291, are very rarely met with, slighter forms of it, like the girl in Fig. 292, are not so very uncommon.

For the somewhat complicated particulars of the developmental defect on which the peculiar conformation of the face and cranium in these children depends, the reader is referred to Mr Greig's interesting paper, in which its details are fully described.

The *clinical features* vary to some extent according to the severity of the case. In the little girl shown in Figs. 289 to 291, who was the illegitimate child of a mentally defective woman,² the mental condition was much below normal. She was a rather low-grade imbecile. In the milder cases also, the intellect is probably always somewhat below the average.

¹ *Edin. Med. Journ.*, Oct. 1924, N.S., xxxi., 560; see also D. C. Muir, *Brit. Journ. Child. Dis.*, April to June 1925, xxii., 102.

² Thomson, *Trans. Med. Chir. Soc. Edin.*, 1904, N.S., xxiii., 208.



FIG. 289.—Hypertelorism. (Mary McD. at 7 years.)

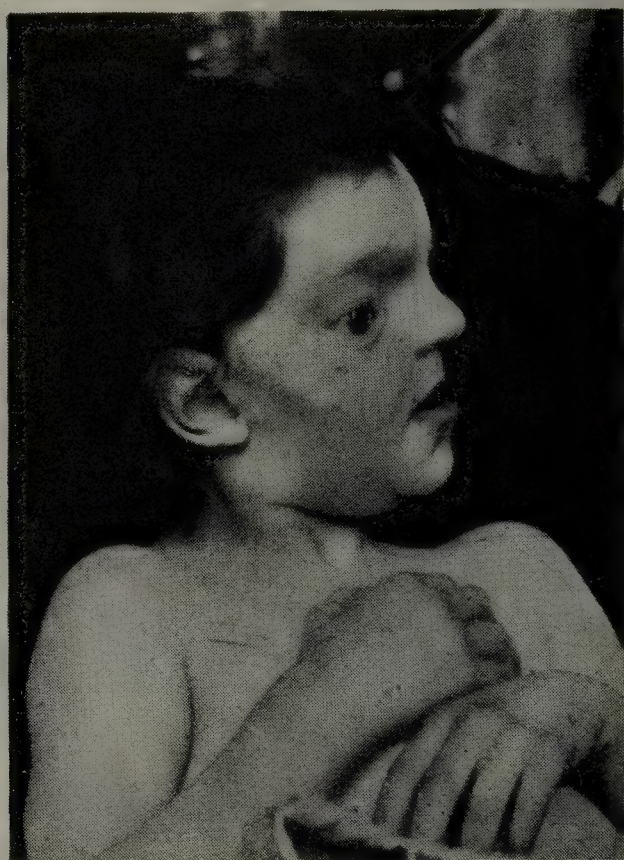


FIG. 290.—Hypertelorism. (Mary McD. at 7 years.)



FIG. 291.—Hypertelorism. (Mary McD. at 19 years.)

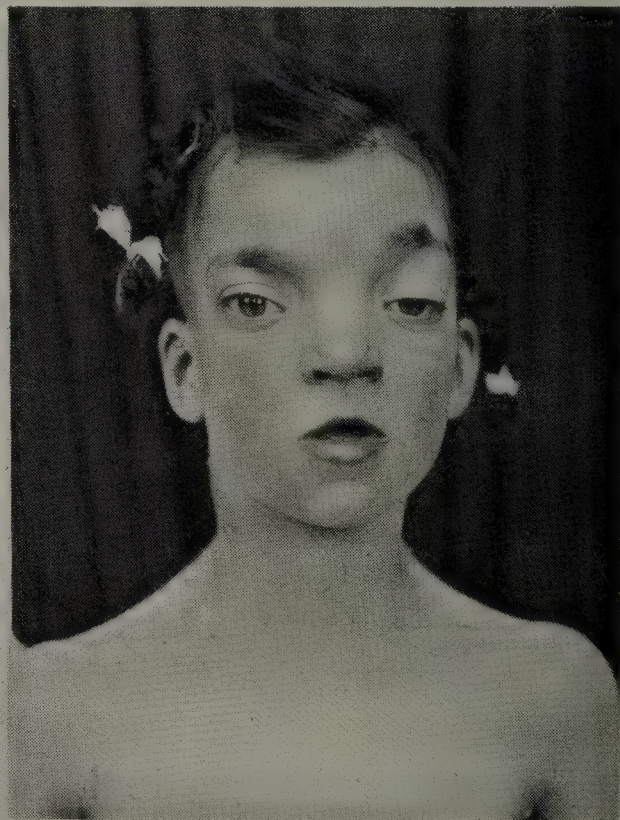


FIG. 292.—Hypertelorism. (Annie C. at 12 years.) (Mr Greig's case.)

The malformation of the face is striking. The forehead is low and broad, with prominent frontal eminences which are separated by a shallow mesial groove. The orbits are large and widely apart, and they diverge so much that binocular vision is impossible for near objects. The child therefore holds the head inclined to one side and looks with one eye at a time, as a bird does. The bridge of the nose is extremely broad, and the nostrils widely open. The upper jaw is abnormally small—especially the premaxillary portion—and the palate, in early childhood, is high and narrow. The mouth is usually kept open. The lower jaw is fairly normal; and the first teeth show no abnormality in form or arrangement. Although the general growth of the body and extremities remains below the normal there is no definite deformity present, except in the face and head. The eyes, heart, and other organs show no structural abnormality.

Multiple Exostoses (*Diaphysial Aclasis*).

Pathology.—The disease which gives rise to multiple exostoses has been shown by A. Keith¹ to be a disorder of growth analogous to achondroplasia, and not a form of tumour-growth as was formerly supposed. He suggests “*Diaphysial Aclasis*” as a suitable name for it.

As was recognised by John Hunter, the shafts of the bones grow in length by a double process. There is not only deposition of new bone in the cartilaginous growth disc at the end of the shaft, but also a “modelling process” by which the new bone there laid down “is pruned, reformed, and incorporated as an intrinsic architectural part of the cylindrical shaft.” Multiple exostoses result from a disorder of growth in which the deposition of new bone goes on, while the modelling process is retarded and aberrant. Those bones which are formed entirely in cartilage—such as the tarsal and carpal bones, the epiphyses of all the long bones, the vertebral bodies, and the sternum—are unaffected; and those formed in membrane only—such as the bones of the cranial vault and face—are also free from the disease. In the limbs the exostoses occur only in connection with the growing ends of the shafts of the long

¹ *Journ. of Anat.* (Cambridge), Jan. and April 1920, liv., Parts II. and III., 101.

bones, where a core of cartilage-formed bone is encased in a sheath of bone formed beneath the periosteum.

The disease is congenital in origin, but definite exostoses are rarely found in infancy or early childhood. The period during which they most often occur is between ten and twenty years, when the growth in the limbs is most active. After the twenty-fifth year, about which time the skeleton ceases to grow, no fresh exostoses appear; and, though those present remain, they may diminish appreciably in size in later life.



FIG. 293.—Congenital Defect of the Patellæ and Nails. (Boy of 6 years.)

The **etiology** of the disease is quite obscure. Keith suspects that it may depend on a disturbance of the function of one or more of the endocrine glands. Certainly neither rickets, syphilis, nor rheumatism has anything to do with its occurrence. The condition is sometimes hereditary, and boys are affected about twice as often as girls.

Clinical Features. —

The exostoses are in themselves painless, but when they are growing rapidly

they may cause distress from pressure on nerves. They should generally be left alone, unless they are causing pain or tenderness or producing deformity and interference with movement, as they are especially apt to do on the fingers. Excellent results may, however, sometimes be obtained by their excision.

Congenital Defect of the Patellæ with Defect of the Nails.

The patella is not infrequently defective or absent in cases of severe congenital club-foot and in some other deformities of the lower limbs.

A rare and interesting hereditary and family form of this defect¹ is occasionally met with, in which a mother and several

¹ A. C. D. Firth, *Brit. Journ. Child. Dis.*, July 1912, ix., 305.

of her children have the patellæ either very small or quite absent, and, along with this, an arrested development of the nails, which on both fingers and toes are small, ill-formed, and concave (Fig. 293). The patients are generally healthy and well-formed in all other respects, except that a few of them may be unable to straighten their elbows completely. In one family under the care of one of us (J. T.) the mother and five children presented defective patellæ and nails, while the father and the other five were normal in every way.

This condition has a special interest and importance from the point of view of normal development, as it proves beyond all doubt, what does not seem ever to have been suggested by embryologists, that a close relationship exists between the development of the patellæ and that of the nails.

The absence of the patellæ does not appear to interfere to any extent with the usefulness of the limbs.

Congenital Elevation of the Scapula (*Sprengel's Deformity*).

Congenital high scapula is a not very rare deformity in which one shoulder-blade is placed higher by an inch or two than the other. The affected bone is nearly always smaller than the other one, and is tilted round so that its angle is nearer the spine than it should be. The deformity results in some restriction of the movements at the shoulder-joint, so that the arm cannot be raised to more than a right angle from the trunk. In about a half of the cases there is congenital scoliosis. In some the vertebral border of the affected scapula is connected with the spine of the sixth or seventh cervical or the first dorsal vertebra by a fibrous band or by a triangular plate of bone or cartilage (Fig. 294). In rare instances both sides are affected.¹

In most of the cases no other defect is present; but, in a considerable minority, there are associated deformities. There may, for example, be a partial absence of several of the ribs in the neighbourhood of the shoulder-blade (Greig), or a congenital pectoral defect (p. 847), or a complete or partial deficiency of some other muscles, or of single vertebræ; and

¹ Willett and Walsham, *Lancet*, 1883, i., 453; Sprengel, *Arch. f. klin. Chirurg.*, 1891, xlii., 545; D. M. Greig, *Edin. Med. Journ.*, 1911, N.S., vi., 242.

quite a large number of other associated defects have been described in a few cases.

Etiology.—The causation of the abnormality is still uncertain, but there can scarcely be any doubt that it is of the same nature as that of congenital unilateral pectoral

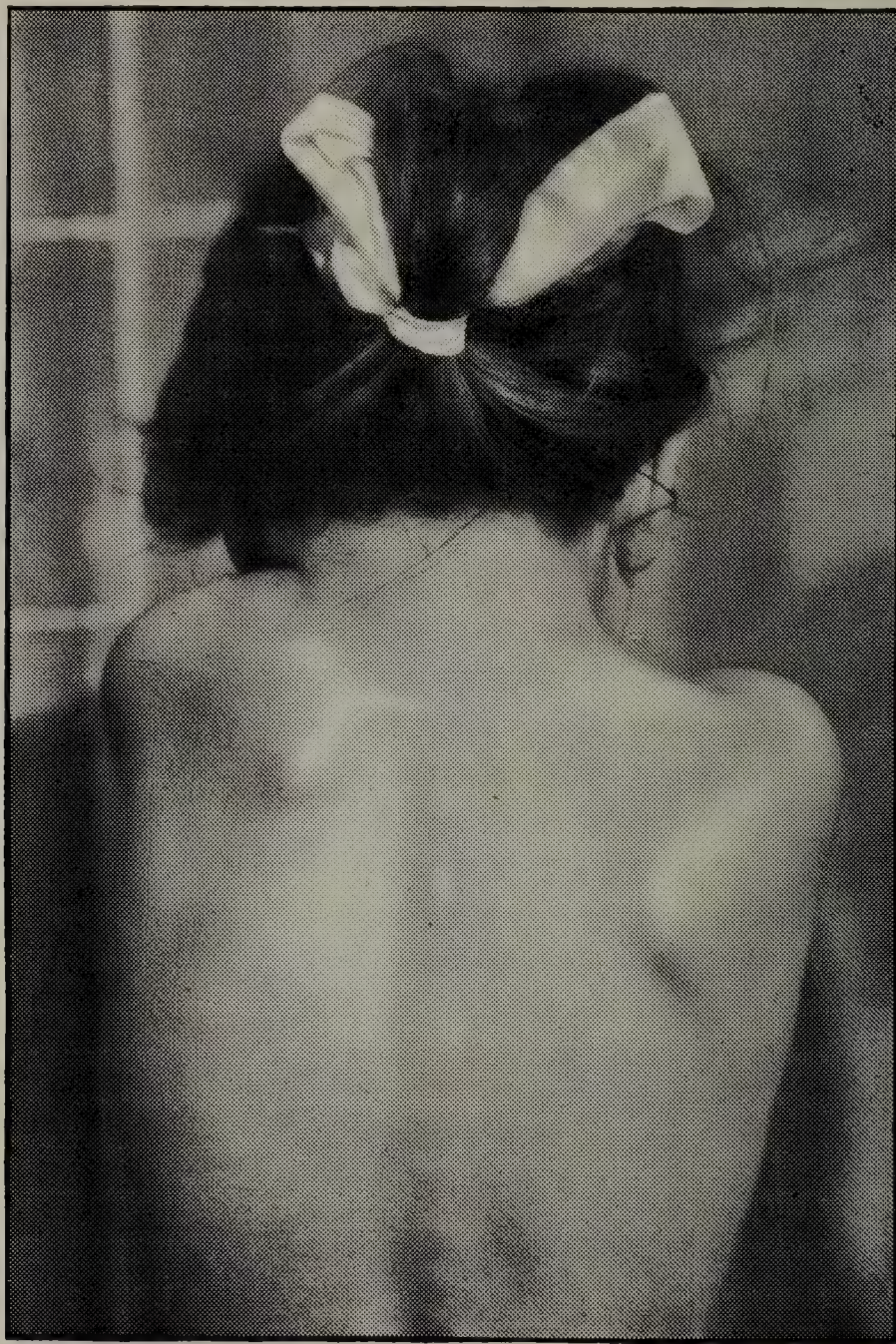


FIG. 294.—Congenital Elevation of the Scapula, with bridge of bone uniting scapula and spine. (Girl of 10½ years.)

defect, and that no theory is satisfactory that does not account for both conditions. A variety of theories have been advanced. That of Sprengel seems to be the most probable. According to this, the malformation is brought about by some kind of abnormal pressure on the shoulder during intra-uterine life, along with prolonged fixation of the arm behind the back; and this pressure is supposed to be

connected with a deficiency of the liquor amnii. In some instances there has been no lack of amniotic fluid at birth, but this does not prove that it may not have been scanty during an earlier stage of the pregnancy.

Treatment.—If there is a bony or other attachment of the scapula to the vertebræ, considerable improvement may result from its removal. Otherwise there is no advantage to be gained from a surgical operation.

CHAPTER XXXV

RHEUMATIC INFECTION IN CHILDHOOD¹

MANY of the older authorities on the diseases of children have written of rheumatism as a rare disease in early life. This is not to be wondered at since for long the disease was considered one principally of the joints, with carditis as a complication. When, however, it became recognised that arthritis during childhood was mild and evanescent, and indeed often absent, that Sydenham's chorea was one of the most frequent manifestations of the disease, and further, that much of the heart disease in adult life was due to rheumatism contracted many years before, then the true importance of the rheumatic infection during childhood, not only on account of its frequency but also because of its gravity, was first visualised. In the bringing about of this change in our outlook the names of Bouillaud, Begbie, Roger, Sée, Barlow, Cheadle, and Osler stand out prominently in a story which took close on one hundred years for its unfolding.

The modern conception of the rheumatic infection is that it is a disease, analogous to tuberculosis, which may persist for months or years in a latent state, but with a marked tendency to declare its presence by the development of acute exacerbations in various susceptible tissues. And, as shown by Begbie in 1847 and repeated by Cheadle in 1897, no one manifestation can be considered the primary lesion. One of the most characteristic features of the disease, at least during childhood, is that the various manifestations, with the exception of nodules, may appear in any order or combination. As Cheadle² says: "Sometimes—perhaps most frequently—arthritis appears first; in other cases endocarditis; now and again chorea inaugurates the morbid series."

Incidence of the Rheumatic Infection.—At the present moment it is not possible to state the true incidence of this

¹ L. Findlay, *Rheumatic Infection in Childhood*, London, 1931.

² W. B. Cheadle, *Allbutt's System of Medicine*, London, 1897, iii., 39.

disease in the country generally, or whether it is increasing in prevalence or is on the wane. Within recent years, however, notification has been instituted in certain metropolitan boroughs (Paddington, Kensington, and Holborn) and in the city of Edinburgh, and although the total number of cases recorded in any one area seems small, and is probably an underestimate, when the case rate per 100,000 children is compared with the case rate of tuberculosis for the same age-period the seriousness of the problem becomes apparent.

*Table showing Notification Rate of Tuberculosis (Pulmonary and Non-Pulmonary) and the Rheumatic Infection in Children per 100,000 Living.**

Place.	Year.	Age-period.	Rheumatic Infection.	Pulmonary Tuberculosis.	Non-Pulmonary Tuberculosis.
Edinburgh .	1931	0 to 14 years	88	55.0	148
Kensington .	1930	0 „ 16 „	239	46.00	36
Paddington .	1930	0 „ 16 „	134	9.4	161
Holborn .	1930	0 „ 15 „	268	14.0	73

* These figures were supplied by the Medical Officers of Health of the various centres.

Etiology.—Although it is generally accepted that the cause of rheumatism is in all probability some microbic factor, its exact nature has not been disclosed. The search for a bacterial factor in this disease has met with the most discordant results. While some workers record invariably negative findings, others have described a diplo- or strepto-coccus which they have isolated from the vegetations on the valves, the pericardial exudate, and the joint effusion, as well as from the subcutaneous nodules. It is quite apparent, however, that these various workers are dealing with different organisms, so much do they vary in their cultural and biological characters.

One has a difficulty in believing that such a specific infection as rheumatism is caused by the greatest variety of organisms, and yet this is the most recent suggestion. The supporters of this view postulate that there is a state of hypersensitiveness (allergy) to streptococci, either congenital in origin or induced by the patient harbouring any of these organisms. It is further

stated that the particular rheumatic hypersensitiveness is not specific, since the various reactions (manifestations of the disease) may be called forth by the greatest variety of strains of streptococci, although the allergic condition had been induced by one particular type of organism. Opinion on the matter must in the meantime be reserved, and it should not be forgotten that perhaps the *contagium vivum* is of the nature of a parasite. The specificity of salicylates suggests such a possibility, since it is only in the case of parasitic diseases that we are in possession of specific remedies.

Whatever the true exciting cause of the disease there are certain definite *predisposing factors*. Of these, social conditions are the most important. Rheumatism is a disease of the hospital-class, and any theory of its etiology must take this into account. While the hospital out-patient departments and wards are never free of examples of the disease, it is rare to meet with it among the children of the well-to-do. It does not appear to be the very poorest of the community which suffer, but the better-class working families.

The housing conditions are often unsatisfactory. Residence in low-lying neighbourhoods, along water-courses, and dampness of the home are strong predisposing factors. The immunity of the same class of child when resident under good hygienic conditions in poor-law schools or institutions is a very suggestive fact.

It is frequently stated that certain types of children are specially prone to the disease, as, *e.g.*, the red-haired and blue-eyed, but no trustworthy statistical study has been published to support this contention. It would seem undoubted, however, that *heredity* is of considerable importance. In our experience a family predisposition was obtained in 35 per cent. of the patients of the hospital-class and in 60 per cent. of those of the well-to-do.

There is no definite evidence that diet plays any part in the etiology of rheumatism.

Previous Disease.—Whatever the portal of entry of the rheumatic virus there would appear no doubt that disease of the tonsils is a predisposing factor. Many examples of the disease set in with an attack of tonsillitis, and diseases such as scarlet fever and diphtheria are specially prone to be followed by rheumatism.

Geographical Distribution.—The disease is specially prevalent in the British Isles, the United States of America, France, and Germany, but it is rare in the eastern states of Europe, in India, in South Africa, and in Australasia.

Seasonal Incidence.—The season of the year exerts an influence on the incidence of rheumatism. In England it is most prevalent during the autumn and winter, and in America during winter and spring. Everywhere it is least common during the summer.

Sex Incidence.—The rheumatic infection attacks girls more commonly than boys. The sex incidence of the various manifestations in a series of 701 cases observed at the Royal Hospital for Sick Children, Glasgow, is shown in the following Table:—

Sex Incidence of Various Rheumatic Manifestations.

Condition.	Males per cent.	Females per cent.	Proportion.	Total.
Total Rheumatic cases . . .	37.0	63.0	1 to 1.6	701
Arthritis (alone) . . .	47.6	52.4	1 „ 1.1	321
Arthritis and Chorea . . .	27.9	72.1	1 „ 2.5	179
Chorea (alone) . . .	26.2	73.8	1 „ 2.8	164
Total Cardiac cases . . .	37.2	62.8	1 „ 1.6	489
Mitral Regurgitation . . .	41.0	59.0	1 „ 1.4	215
Mitral Stenosis (pure) . . .	41.6	58.4	1 „ 1.4	12
Aortic Disease . . .	49.2	50.8	1 „ 1.0	59
Pericarditis . . .	34.7	65.3	1 „ 1.8	69
Rheumatic Nodules . . .	42.4	57.6	1 „ 1.3	73

Age Incidence.—The various manifestations of the rheumatic infection have the same age incidence. This is in keeping with our previous remark that no one is of the nature of the primary lesion. It is generally stated that the disease never occurs during the first year of life, and only on very rare occasions before the age of two years. The age of maximum incidence would appear to be between seven and eight years.

Manifestations of Rheumatism.—The chief manifestations of rheumatism are (1) arthritis, (2) chorea, (3) carditis embracing endocarditis, pericarditis, and myocarditis, and (4) subcutaneous nodules.

Arthritis.—Arthritis in childhood is characterised by its general mildness. As Cheadle¹ remarks, “In the rheumatism

¹ W. B. Cheadle, *Allbutt's System of Medicine*, London, 1897, iii., 38.

of childhood arthritis is at its minimum." Usually it is of a subacute nature, tending to be evanescent and to undergo spontaneous recovery. It is because of this feature that the condition is so often mistaken for growing pains. The arthritic manifestations may be, however, exceedingly severe, with excruciating pain, great swelling and tenderness, and redness and œdema of the overlying skin. Fever and increase in pulse-rate are more or less parallel with the acuteness of the mischief. The temperature is rarely above 103° F. and usually runs between 102° F. and 103° F. Hyperpyrexia is exceedingly rare.

It is generally stated that the smaller joints of the hands and feet are affected in the case of the child, but in our experience the arthritis has most frequently involved the wrists, ankles, knees, and shoulders. The vertebral joints usually escape. As in the adult, the mischief is apt to flit about, one joint recovering as another is attacked, although many joints may be involved at one time.

The onset is usually sudden, with fever and general malaise in the midst of good health. Pain and swelling of the joint may develop synchronously with the fever, but at times the arthritis may not appear for two or three days, and thus at first the malady may be mistaken for influenza. Tonsillitis may precede the attack of arthritis, or it may be ushered in by severe epistaxis. Occasionally the onset is slow, and there may be complaint of generalised pain and stiffness of the limbs for some time before the true nature of the disease declares itself. In still other cases the mischief may be preceded by a period of ill-health, characterised by lassitude, nervousness, and intermittent limb pains.

In contrast to what prevails in the adult, perspiration, unless during the administration of salicylates, is not a prominent feature of the disease in the child.

Pathologically the condition is an inflammation of the synovial membrane and surrounding tissues, with effusion into the joint. This latter varies in appearance from that of a glairy straw-coloured fluid to that of frank pus depending on its cellular content, which, on microscopic examination, is revealed to consist of polymorphs. In our experience the effusion into the joint has invariably been sterile.

Chorea.—The association of chorea with rheumatism was first noted by Bouteille¹ in 1809. At a later date (1821)

¹ Bouteille, *Traité de la chorée ondance de St Guy*, Paris, 1809.

Copeland¹ drew attention to the alternation of chorea and arthritis in a boy of nine years, and in 1839 Bright² described the association between chorea and pericarditis, which was at that time recognised as due to rheumatism. Bright thought that it was the pericarditis by reflex action through the phrenic nerve which caused the chorea. It was Begbie³ of Edinburgh who, in 1847, first appreciated the true relationship of chorea in the rheumatic series, and stated that chorea, arthritis, and carditis, jointly and severally, all originated in the same specific disorder. This teaching, which is the view held to-day, only very slowly, however, gained general acceptance.

It must be clearly understood that chorea is only a symptom, and may be called forth by different infective agents, and hence is no more invariably rheumatic in origin than either arthritis or carditis. But apart from encephalitis lethargica, which within recent years has been a frequent cause, and syphilis, which is occasionally the etiological factor, the rheumatic virus is the common cause.

The nature of the lesion in chorea is not known. The absence of any abnormality of the cerebro-spinal fluid, as well as of any mental deterioration, speaks against any meningo-encephalitic mischief. From analogy with encephalitis lethargica the lesion in all probability is situated in the basal ganglia, but in the few cases examined within recent years nothing more than degeneration of nerve cells has been observed.

In contrast to the condition as met with in encephalitis lethargica the rheumatic variety is insidious in onset. It may, however, be ushered in by an attack of tonsillitis, be preceded by a period of ill-health with indefinite pains and lassitude, a certain degree of nervousness and a tendency to enuresis. The first evidence noticed may be a tendency to grimace or to shuffle with the feet while standing at attention, and hence at school it is sometimes mistaken for naughtiness. There may be twitching of the arm so that writing is interfered with, or the child may spill his food while feeding himself. These features are usually accompanied by increased emotionalism, so that from little cause the child bursts into tears. Mentally, however, the child seems quite bright; in fact, during the acute stage he reacts

¹ J. Copeland, *London Medical Repository*, London, 1821, xv., 23.

² R. Bright, *Trans. Medico. Chir. Soc.*, London, 1839, xxii., 1.

³ J. Begbie, *Med. Chir. Soc.*, Edin., 17th Feb. 1847.

on the average better to mental tests. Occasionally there is a degree of mental dullness and loss of memory with a curious mask-like appearance of the face (Figs. 295 and 296).

Gradually the involuntary movements affect more and more of the body, and increase in severity until by the end of a week or ten days the child is constantly twisting and turning, and may require to be protected against injuring himself. Speech becomes affected, as also deglutition, and feeding may be a matter of extreme difficulty. The choreiform movements do not always become generalised, but may remain limited to one limb or to one side of the body (hemi-chorea). Accompanying the movements there is marked hypotonus and even weakness of the limbs affected. This weakness on occasion is extreme and may amount to paralysis (paralytic chorea), when for a time all movements cease; they return, however, as the paralysis passes off.

Uncomplicated chorea is unaccompanied by fever, so that a rise of temperature either should raise doubt regarding its rheumatic nature, or suggest the development of some other rheumatic manifestation (arthritis, endocarditis, or pericarditis). It is worthy of record that if some other rheumatic manifestation develops the chorea is apt to disappear. This is specially noticeable if arthritis should supervene, but is also the case if pericarditis ensues. It was because of this behaviour that the older writers spoke of arthritis and chorea being antagonistic.

Chorea for the most part is a self-limited disease, and runs a course of between six and ten weeks. This is an important point to bear in mind when assessing the value of any therapeutic measure. Like other rheumatic manifestations chorea shows a marked tendency to relapse, and some children may suffer from as many as eight or nine attacks. It is interesting to note, just as is the case with arthritis, that when the rheumatic series is introduced with chorea, recurrences tend to be also of the nature of chorea.

Chorea shows the same tendency as arthritis to appear during the winter months, and to be least prevalent during the summer. Of all the manifestations of rheumatism, it is the one which shows the greatest predilection for females, the proportion of boys to girls being 1 : 2.8 (p. 897).

In a proportion of the examples of chorea some other rheumatic manifestation appears. Some 50 per cent. suffer in



FIG. 295.—Physiognomy of Chorea at onset of attack.



FIG. 296.—Same patient after convalescence.

(Dr J. S. Fowler's case.)

addition from arthritis, and about the same proportion develop a cardiac lesion. It is, however, the minority (about 24 per cent.) of rheumatic children who suffer from both arthritis and chorea. The majority present only arthritis or chorea in addition to carditis. This raises the question of there being two strains of the rheumatic virus. Moreover, when carditis does supervene in a child who has had no other manifestation than chorea, it is almost invariably a lesion of the mitral valve with a marked tendency to proceed to stenosis. Aortic disease and pericarditis, but particularly pericarditis, and also the subcutaneous nodule, seldom occur in a patient who has only suffered from chorea.

Carditis.—The term “carditis,” which embraces endocarditis, pericarditis, and myocarditis, has the value that it focuses attention on the myocardium, which of all the structures is most frequently involved, and which is of such significance for the function of the organ. But it is wise that it be not employed to the exclusion of the particular valvular or other type of lesion, as this has the greatest bearing on prognosis.

It is the cardiac manifestations which give the rheumatic infection its gravity, and prognosis undoubtedly depends on their presence and nature. Implication of the heart may not reveal its presence by any sign or symptom unless when well advanced. This is its great danger. There is usually some increased frequency of the pulse, but so easily is the child's pulse upset that it is difficult to assess the value of this sign. Unless in the case of pericarditis there may be no rise of temperature.

Although carditis as a rule follows or is synchronous with an attack of either arthritis or chorea, it may be the first manifestation of the series to appear, or even the only evidence of the disease. This was so in 7 per cent. of the examples observed at the Royal Hospital for Sick Children, Glasgow.

It is characteristic of childhood that the *myocardium* is often the part chiefly affected. If the lesion is mild in degree, only a slight temporary dilatation occurs, with increase of the dullness to the left, accentuation of the pulmonary second sound, and some impurity of the first sound or a slight systolic murmur at the apex. If severe, serious and prolonged dilatation may result. A large proportion of the loud purely systolic mitral murmurs, which follow rheumatic attacks in children, recover in time completely and are probably due to myocardial affection and not to valvular disease.

Endocarditis, usually of the mitral valve, is also common. It is probably entirely recovered from in some cases, but it often leads to permanent valvular defect, with more or less compensatory hypertrophy. Mitral regurgitation is the commonest variety of lesion. Stenosis, however, is not infrequently observed. This may occur comparatively early during the course of the illness, and often first declares its presence by a third sound or diastolic roll audible at the apex. The aortic valve is much less frequently affected, probably in less than 50 per cent. of the cases. The diastolic murmur characteristic of aortic regurgitation is often first heard at the pulmonic area. The water-hammer pulse, capillary pulsation, and pulsation of the retinal vessels are late phenomena and seldom met with during childhood.

As previously mentioned, mitral disease, and especially mitral stenosis, is more related to the choreic type of rheumatism and aortic disease to the arthritic type.

Pericarditis is the least frequent of the cardiac manifestations, but would appear to be more common in the rheumatism of childhood than in that of the adult. Like the other types of carditis, inflammation of the pericardium as a rule follows some other manifestation, but it may be the first evidence of the infection. In one-third of the examples observed at the R.H.S.C., Glasgow, pericarditis was either the first manifestation or developed during the earliest stage of the disease. Recurrent attacks of pericarditis are not uncommon. As previously mentioned, pericarditis seldom occurs in association with chorea.

The onset of pericarditis is often insidious, and is apt to be overlooked unless one is on his guard. A rise in temperature and complaint of sub-sternal pain often direct attention, as also cyanosis and dyspnoea, but not infrequently the condition is discovered accidentally during a routine physical examination. Hence it should be a rule to keep a constant watch on the heart during any active phase of the disease (p. 593).

In addition to the increased area of cardiac dullness upwards, as well as to the right and to the left, dullness to percussion at the left base behind is not uncommon. This has been variously ascribed to collapse of the lung from pressure by the distended pericardium, to the distended pericardium itself extending posteriorly and displacing the lung, and to a so-called rheumatic pneumonia, but in our experience it is caused by a passive

effusion as verified by exploratory puncture, and finding in the sediment plaques of endothelium which are characteristic of a transudate.¹

*Rheumatic nodules*² are an extremely important manifesta-



FIG. 297.—Rheumatic Nodules on Elbow.

tion of the disease from a diagnostic and prognostic point of view. In children, for all practical purposes, they not only indicate the presence of rheumatism, but show that the disease is present in a serious and progressive form, and that active antirheumatic treatment should at once be adopted. They are to be found in a considerable

proportion of rheumatic attacks in children in this country, but are said to be less common on the Continent and in some parts of America. In very rare instances they are present in rheumatoid arthritis, and as the only abnormality in definitely non-rheumatic subjects.³

The nodules vary from the size of a pin's-head to that of a pea, or even larger (Figs. 297 to 300). Generally, only a few are found at a time, usually over the bony prominences of the elbows, knees, or ankles, but occasionally they are present in large numbers, growing not only about all the bony prominences of the limbs, over the vertebral



FIG. 298.—Rheumatic Nodules on Knee.

¹ L. Findlay, *Arch. Dis. Child.*, 1930, v., 259.

² Barlow and Warner, *Trans. Seventh Internat. Med. Congress*, London, 1881, iv., 116; Poynton and Still, *Trans. Path. Soc. Lond.*, 1889, 39; C. Coombs, *Journ. Path. and Bacteriol.*, 1910, xv., 489; Bronson, Carr, and Perkins, *Amer. Journ. Med. Sci.*, June 1923, No. 6, cxlv., 781; C. O. Hawthorne, *Studies in Clinical Medicine*, London, 1912.

³ L. Findlay, *Rheumatic Infection in Childhood*, London, 1931, p. 104.

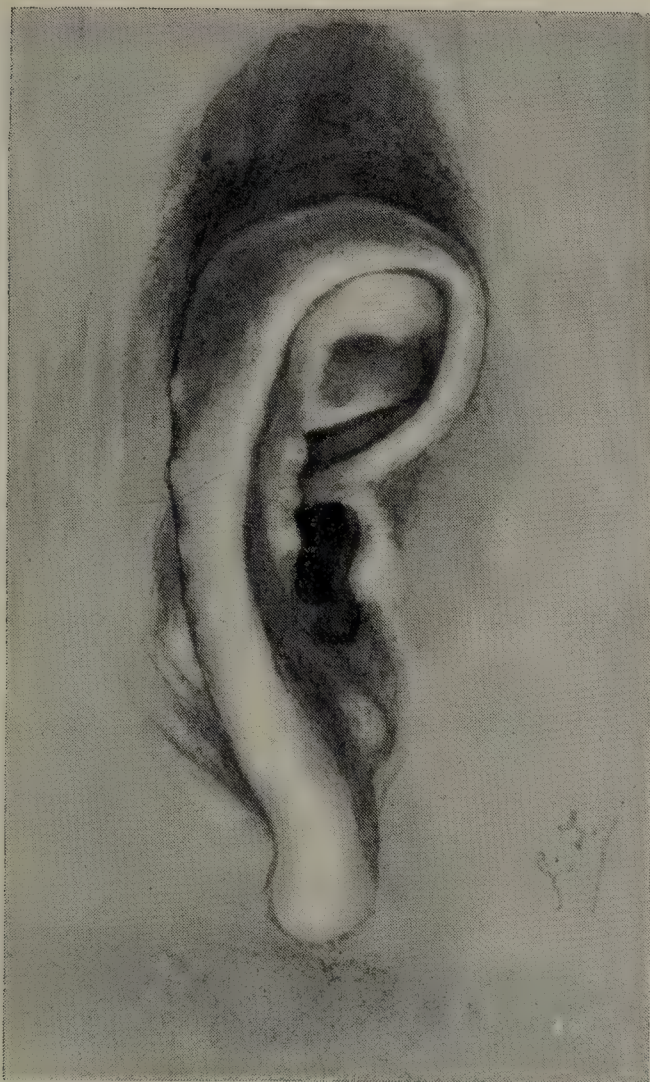


FIG. 299.—Rheumatic Nodules on Auricle.



FIG. 300.—Rheumatism and Chorea. Hair cut to show Nodules on Scalp.

spines and under the scalp, but also on the ribs, clavicles, scapulæ, and iliac crests and over the prominent tendons of the extremities, and the fasciæ of the erector spinæ and abdominal muscles—even, rarely, on the rims of the ear (Fig. 299). They appear in crops lasting usually a few weeks, sometimes several months, and rarely more than a year; but we have, in one instance, watched several for more than eighteen months before they disappeared. Crops of nodules may recur annually for several years. When they occur in adults they sometimes last much longer; in one case, a man over forty, large typical nodules lasted for many years. The skin over the nodules is not reddened, and is not closely adherent to them. They are somewhat loosely attached to the periosteum or any other fibrous membrane over which they lie; and are not at all painful or tender, except to a slight degree, when they are growing rapidly in tense structures, such as the scalp or palm, or have been irritated by pressure. When present, they are easily recognised if looked for; and if the skin is moved over them in a good light, they are even more easily seen than felt. Occasionally the nodules are present in large numbers, as in the case represented in Figs. 299 and 300, in which more than two hundred were counted at one time in different parts of the body.

The subcutaneous nodule is of an inflammatory nature, being composed of fibrous tissue, in different stages of development and different degrees of cellularity, arranged around new-formed vessels, and at times may show the picture of giant-cell formation as met with in the Aschoff body.

Tonsillitis, although not invariably or even usually a rheumatic manifestation, is not infrequently associated with the disease. Haygarth¹ as long ago as 1805 stated that people with sore throats are more subject to rheumatism. The tonsil is considered the chief portal of entry of the *contagium vivum*, and not uncommonly an attack of arthritis or chorea is preceded by one of tonsillitis. Tonsillectomy during the course of the rheumatic infection may cause an acute exacerbation, and there would appear no doubt that it is because of disease of the tonsils in scarlet fever that some manifestation of the rheumatic infection is such a frequent sequel to that exanthem. It would also appear that a preliminary tonsillectomy renders the

¹ J. Haygarth, *A Clinical History of Acute Rheumatism*, London, 1805.

individual less susceptible to arthritis, and to carditis when arthritis does supervene, but there is no evidence that the operation has any effect on the incidence of chorea, which again raises the question previously referred to of there being two strains (an arthrotropic and a neurotropic) of the rheumatic virus.

Certain skin conditions, *e.g.*, *erythema multiforme*, *psoriasis*, and *purpura*, may occasionally have a rheumatic basis, but this cannot be recognised unless their development is synchronous with some other undoubted rheumatic manifestation. *Erythema nodosum*, at one time generally considered a rheumatic manifestation, is no longer believed to be so, but to be due to streptococci and the tubercle bacillus (p. 391).

Pneumonia, *pleurisy*, *peritonitis*, and *meningitis* are credited by some writers with being at times of a rheumatic nature. Naish¹ and Fraser² have recently described pneumonic consolidation in patients with severe rheumatic cardiac disease, and because of the presence of cellular conformations simulating Aschoff's bodies they look upon the lesion as truly rheumatic.

Most writers believe that the rheumatic toxin plays a part in the causation of idiopathic pleurisy. In our experience idiopathic pleurisy seldom occurs in a rheumatic subject and has never synchronised with one of the definite rheumatic manifestations. Effusion into the pleura is not uncommon as a complication of pericarditis, but, like that developing in consequence of cardiac failure from any cause, is of the nature of a transudate, as proved by the character of its cellular contents (p. 904).

Many writers state that the rheumatic toxin may cause peritonitis,³ or even appendicitis,⁴ but their records leave much to be desired. Abdominal pain may be present in any acute illness such as acute rheumatism. It must also be remembered that the pain may be referred from the pericardium in inflammation of the sac, and that confusion may arise between mischief in the abdomen and in one or other hip-joint. When the right hip-joint is affected, appendicitis is apt to be diagnosed.

¹ A. E. Naish, *Lancet*, 1928, ii., 10.

² A. D. Fraser, *Lancet*, 1930, i., 70.

³ J. R. Paul, *Bulletin Ayer Clin. Laby. Penn. Hosp.*, Philadelphia, June 1930, p. 17.

⁴ J. J. J. Giraldi, *Archiv. Dis. Child.*, London, 1930, v., 379.

Recent work has shown that during the acute stage of arthritis or chorea there may be some impairment of *hepatic function* as well as that due to passive congestion consequent on cardiac decompensation.

A certain degree of *anæmia* of chlorotic type is not uncommon, but so far as one can judge it has no relation to the severity of the mischief or to the presence or otherwise of cardiac disease.

There is no authentic case on record of rheumatic *meningitis*. This manifestation is a relic of pre-bacteriology days, when septicæmia and rheumatism were frequently confused.

Diagnosis.—There is no doubt that much is diagnosed as rheumatism which is not of any such nature. For this tendency the description of the “pre-rheumatic state” is in great part responsible. Indefinite muscular pains, general malaise, habit-spasms, recurrent sore throats, are often erroneously considered evidence of the disease. Undoubtedly the tendency for the arthritis to be mild and evanescent makes the question of diagnosis one of no little difficulty. In true rheumatism the pains are as a rule specially severe for a day or so, and only troublesome at more or less long intervals, and thus point to the cause being a subacute attack of arthritis. On occasion the early stage of rheumatoid arthritis, or a syphilitic or gonorrhœal arthritis, may be mistaken for rheumatism, but the absence of any immediate response to full doses of salicylates should at once make the matter clear.

In tuberculosis a subacute polyarthritis (*rhumatisme tuberculeux*) may develop and simulate rheumatic arthritis, and, when pericarditis also occurs, the differential diagnosis may be a matter of some difficulty. According to the French,¹ this association of arthritis with tuberculosis is a frequent occurrence, and although there is no doubt that such a syndrome is met with, it is, in our experience, decidedly rare.²

Habit-spasm bears no true resemblance to chorea. In habit spasm the contraction is always of the same nature and of the same muscle or group of muscles. The condition may spread from one muscle to neighbouring groups, but it never, as chorea, involves the whole body. Chorea may also occur in syphilis and in encephalitis lethargica. In the latter infection it is acute in onset and reaches a height within twelve or twenty-four hours,

¹ A. Poncet and R. Leriche, *La Tuberculose Inflammatoire*, Paris, 1912, 122.

² L. Findlay, *Glasg. Med. Journ.*, 1920, N.S., xciii., 241.

whereas in rheumatic chorea the development is slow and does not reach its maximum for a matter of ten days or so.

Prognosis.—The prognosis in rheumatism is a most difficult matter. It depends primarily, as already mentioned, on the presence of cardiac disease and on the particular type of cardiac lesion which has developed.

So far as danger to life is concerned, pericarditis is the most grave variety of carditis, probably more because it implies a widespread myocarditis than because of any mechanical embarrassment. Many, probably as high a proportion as 50 per cent., die during the acute phase of the disease, and of those who get over the acute illness the vast majority suffer from severe cardiac disability. Aortic disease is next in order of gravity, both as regards immediate and remote prognosis. Mitral regurgitation, even when accompanied by stenosis, is compatible with long life, and for many years may cause only very slight disability. It should be borne in mind that the presence of a mitral systolic murmur is not always evidence of permanent valvular disease, or even of valvular disease at all. During any acute phase of the mischief (arthritis or chorea), in consequence of the accompanying myocarditis, a temporary relative incompetency of the mitral valve may ensue. When the myocarditis subsides the valvular ring returns to its normal dimensions. It is also possible that a true valvulitis may be recovered from. Whatever the explanation, mitral systolic murmurs often disappear, less frequently mitral pre-systolic murmurs, but it is worthy of note that when once an aortic regurgitant murmur appears this is invariably permanent.

It is worth remembering that carditis usually supervenes prior to, or during the first or second attack of arthritis or chorea, and hence if a child has passed through two attacks of the mischief, counting chorea and arthritis as an attack, with the heart unscathed, he is unlikely to suffer from carditis, however many attacks he is subject to. This would seem to indicate that whether or not the heart is affected depends on the individual susceptibility of the cardiac tissues.

As previously mentioned, the development of the subcutaneous nodule is of grave prognostic omen. Whether the nodule indicates a susceptible cardiac tissue, or whether it means a specially virulent type of infection, the fact remains that its presence almost invariably denotes a severe and progressive

carditis. But there are exceptions to this rule, and occasionally all evidence of carditis even in the presence of nodules disappears.

The *age at onset* of the disease is a factor of some prognostic significance. The younger the child when the disease begins the more likely is cardiac disease, and severe cardiac disease, to follow. The *length of time* the disease has been in progress is also of significance. The longer the child has been affected, and even in the presence of cardiac disease, the greater chance has he of reaching adolescence or adult life. One of the most striking features of the rheumatic infection in childhood is that when a fatal result ensues it is most likely to do so early in the course of the disease. Of a series of 154 fatal examples of the rheumatic infection observed in the R.H.S.C., Glasgow, in 37·4 per cent. death occurred within one year of onset, and in 77·4 per cent. within five years of the onset of the infection. In most cases death had occurred before the children had reached the age of fifteen years. It would seem that if this critical period—pre-puberty and puberty ages—is successfully weathered, the child will in all probability be spared till between thirty-five and forty-five years of age, which is the age-period when most rheumatic-cardiac deaths occur, no doubt because this is the age-period when arterio-sclerosis makes its appearance.

Treatment.—This may be divided into prophylaxis and the cure and after-care of the infected child.

In view of the tendency for the disease to occur in damp and low-lying houses, and especially if these are situated in the course of water-ways, and because the disease is one which attacks principally the children of the less-favoured classes, one of our chief *prophylactic* measures is suitable housing for the working classes. When we also remember that the tonsil is a frequent, although it is not the only portal of entry of the infective agent, attention to the throat should receive special attention during the early years of life. Apart altogether from any effect which such care has on the incidence of rheumatism, it will increase the child's resistance to disease by improving his general health, as well as minimising his liability to otitis media and cervical adenitis.

At the present moment there is an inclination to ascribe to diet some part in the etiology of the disease. So far nothing of a definite nature has been discovered, so there are no

indications for a special dietary. It is not even certain that under-feeding plays a part, but on general principles, because inanition lowers resistance, this should be guarded against.

Curative Treatment.—Fortunately in the case of arthritis we are in possession of a veritable specific. Salicylate of soda, if given in sufficient dosage, almost, if not invariably, causes an immediate relief of pain, an early decline in the temperature, and a steady diminution in the swelling. Indeed, so constantly do these phenomena result from the presentation of the drug that their non-appearance means either that the arthritis is not of a rheumatic nature, or that endocarditis or pericarditis is also present.

The dose of salicylate should be at least 60 to 90 or even 120 grains per day, and should always be combined with double the amount of bicarbonate of soda, or acidosis may develop. Daily evacuation of the bowels also makes acidosis less likely (p. 565). Salicin, salol, and aspirin are recommended by different writers, but none of these has any advantages over salicylate of soda, and some of them are definitely less efficient. Whatever the drug employed, it should be kept up for some considerable time—in our opinion for three months—even although the arthritis has disappeared, and then presented for periods of two months with intervals of one month for as long as one year.

In addition to the drug treatment the child should be put to bed between blankets, and if the joints are acutely swollen these should be swathed in flannel or cotton wool. The diet during the acute phase should be one of milk and farinaceous food.

No drug except a definite sedative like chloral hydrate, or one of the preparations of bromide, has any effect on the course of chorea, which, as we have previously remarked, extends for a period of six to ten weeks. Since, however, we believe that it is a manifestation of rheumatism, and hence there is a liability to the development of some other manifestation of the disease, we are in the habit of treating chorea with the same therapeutic measures as we do arthritis, *i.e.*, salicylate of soda in large doses combined with alkali. Other drugs, *e.g.*, arsenic *per os* and intravenously, antipyrine, and nirvanol are all recommended, but none has any definite effect on the course of the disease, and they all have the disadvantage that they have not the specific anti-rheumatic effect possessed by the salicylates. Some writers

recommend isolation. Undoubtedly choreic patients are much quieter when isolated, but to practise this as the sole method of treatment is, in our opinion, wrong, since it deprives the patient of the benefit of salicylates.

In the case of any acute manifestation there is no more important factor than the thoroughness with which the treatment is carried out. This depends on the dose of the salicylates given, on the duration of their administration, and on the length of time the child is kept at rest. It cannot be too well appreciated that it is only in this way that involvement of the heart, the really serious event, can be influenced, and hence as much, if not more, attention should be paid to the mild case without carditis as to the patient with the heart already affected. In our opinion the child with either arthritis or chorea should be kept in bed for at least three months, even if the heart is free and the temperature and pulse-rate are normal. In Glasgow it was found that while 80 per cent. of examples of arthritis inefficiently treated during their first attack developed carditis, only 40 per cent. of those treated as above described with large doses of salicylates and confinement to bed for three months suffered from a cardiac lesion.

If there is at the end of three months still increase in the pulse-rate, or fever, the period of complete rest may require to be prolonged. But as the pulse-rate in childhood is apt to be erratic, and occasional fever is not uncommon, it is at times a matter of extreme difficulty to know when the patient should be allowed up. Unless the pulse is unduly rapid, the fever definite, and examination of the heart suggests from increasing size and modification of murmur some activity, we give the patient a certain amount of freedom by allowing him up for half an hour per day and observing the pulse before and after. If the pulse quietens down within fifteen to twenty minutes after returning to bed, the amount of liberty is gradually increased by half an hour per day. Some reliable test for activity of carditis is much needed, and Schlesinger¹ has recently suggested that in the difference between the pulse-rate while awake (the alert pulse) and that during sleep (the sleeping pulse) we may have such an indication. Schlesinger states that in health there is a difference of ten beats per minute, whereas in the presence of carditis this difference is diminished or disappears. The leucocyte count, at

¹ B. Schlesinger, *Quart. Journ. Med.*, 1932, xxv., 67.

one time considered of value in this direction, has been shown to afford no real indication as to the state of the mischief.

The modern conception of the rheumatic infection in that it is a chronic disease like tuberculosis, and requires a long time for its eradication, has been referred to. It was this view which suggested the need for after-care of the rheumatic subject, which was first impressed on the medical profession by Hawthorne¹ and Poynton² in this country. This means the placing of the child who has suffered from a recent active manifestation under ideal conditions where his activity may be graded to his capacity. For this purpose there have arisen "rheumatism supervisory centres" in most towns and so-called "cardiac hospitals" in the country. By this means it is not hoped to cure cardiac disease already developed, but to arrest its progress by diminishing the tendency to relapses which invariably increase its ravages. It is, however, as yet too soon to estimate the real value of these measures.

¹ C. O. Hawthorne, *Trans. Soc. Study of Disease in Children*, London, 18th Jan. 1907.

² F. J. Poynton, *School Hygiene*, London, 1912, III, 131.

CHAPTER XXXVI

SYPHILIS, CONGENITAL AND ACQUIRED

SYPHILIS in children may be congenital, that is to say, it may arise from intra-uterine infection; or it may be acquired after birth. In the former case it is a blood infection from the beginning and there is an absence of the primary sore, the first manifestations being comparable with the secondaries in the acquired form of the disease. Acquired syphilis in the child differs little from the condition as seen in later life, except that the primary sore is usually extra-genital. The manifestations of congenital syphilis, however, in addition to the absence of the primary sore, contrast with those of acquired syphilis in the frequency of snuffles and keratitis, and in its very low degree of contagiousness. Contraction of the disease from an example of the congenital variety is one of the greatest rarities.

Congenital Syphilis.¹

Mode of Infection.—Experience with Wassermann's reaction has shown that the mother of a child with congenital syphilis is almost certainly always herself syphilitic. There is no proof that a father can infect the child *in utero* without infecting the mother also.

Neisser² has demonstrated, by experiments on animals, that the character of the symptoms which follow infection with syphilis depends to a large extent on the site of the inoculation. He found it difficult to convey the disease by subcutaneous injection; but, when he succeeded in doing so, the symptoms were those of a toxæmia. Probably, therefore, the fact of the mother's infection occurring through the uterus may explain

¹ Diday, *Infantile Syphilis*, New Sydenham Soc. Transl., 1861; Hensch, *Lectures on Children's Diseases*, 4th edit., New Sydenham Soc. Transl., 1889, vol. i., 92; J. A. Coutts, *Some Aspects of Infantile Syphilis*, London, 1897; Leonard Findlay, *Syphilis in Childhood*, London, 1919.

² *Beiträge z. Pathol. u. Therapie der Syphilis*, Berlin, 1911.

the "latency" of the symptoms which is observed in so many cases and is so different from what occurs when the virus enters by the skin or vulva. It is probable that Colles' Law is to be explained on these lines. Exceptions to it are extremely rare, and they may be assumed to be examples of superinfection—a second infection having taken place during the course of the first.¹ Many mothers who have shown no primary or secondary symptoms suffer later from tertiary manifestations.

Theoretically it would seem possible that the mother's first infection may sometimes be contracted from a child in her uterus who has received the disease from the father; but, in succeeding pregnancies, she will transmit syphilis to her children. The spirochæte has not yet been demonstrated in semen, but apes have been infected by the seminal fluid of syphilitic men. Undoubted instances have been reported in which one twin was healthy and gave a negative Wassermann reaction, while the other showed typical evidence of congenital syphilis. This is no doubt due to the fact that infection frequently takes place during parturition by means of emboli from the placenta. In this way the passage of emboli along one cord is quite understandable. Moreover the absence of this occurrence (embolism during parturition) readily accounts for definitely syphilitic mothers giving birth to non-syphilitic infants.

There is no undoubted example on record of transmission of syphilis to the third generation. In most of the recorded instances there has been the strong probability of the suspected parent having acquired the disease after the congenital infection has died out.

Infectivity.—The degree of the infectiveness of the spirochæte seems to be usually—perhaps always—destroyed by its sojourn in the infant's tissues.

It has been a common experience in dispensary practice to see healthy children fondling and kissing babies with typical congenital syphilitic eruptions, discharges, and sores, which are now known to contain the organism in great abundance. Yet even the most experienced physicians to Children's Hospitals seem never to have seen any well-authenticated cases in which this practice has led to infection. The few cases on record in which this seemed to have occurred were all possibly explicable in other ways. One of us (J. T.) has known of a number of

¹ Rietschel, *Zeitsch. f. Kinderheilk.*, 1911, Ref. Bd., iii., 577.

healthy infants being vaccinated by mistake from a baby with congenital syphilis, and none of them took the disease. Coutts records a few instances in which adults were apparently infected from syphilitic babies, but it seems impossible to be quite sure that the syphilis might not have been acquired and not congenital. Until the question has been thoroughly investigated, however, it will be well to continue to take precautions.

Symptoms.

The clinical manifestations of congenital syphilis may be divided into three groups, according as they are (1) Early—beginning between birth and two years; (2) Intermediate—between the second and fifth or sixth year; or (3) Late—occurring during or after the period of the second dentition.

1. Early Manifestations.—In early infancy the condition is a blood infection, and its symptoms are mostly analogous to those in the secondary stage of the acquired disease, although visceral lesions, such as only occur in the tertiary period in acquired syphilis, are commonly found.

In the great majority of cases, infants who are the subjects of congenital syphilis are born without any obvious indication of the disease. The presence of snuffling breathing may indeed give grounds for a diagnosis from the very first; but generally for three or four weeks at least one cannot recognise, by clinical examination alone, that they are other than healthy children.

(a) *General Symptoms* (Marasmus, Fever, Irritability).—Although most syphilitic babies seem well nourished to begin with, in some instances *wasting* and debility are noticed in the early weeks. This is often due to the presence of visceral disease, but occasionally it is caused by less serious conditions and responds at once to specific treatment.

When *fever* during the early stage occurs it is generally attributed to the presence of complications from pyogenic organisms; there is, however, no doubt that it is sometimes due to the action of the spirochætes. Great *irritability*, especially during the night, is a common and characteristic occurrence.

(b) *Snuffles and Rash.*—The first definite symptoms to appear are nearly always nasal obstruction, snuffling breathing, and a rash.

The *snuffling* is generally noticed first. It is often present

at birth, and occasionally persists for a long time after the other symptoms have gone. It is sometimes absent, but probably in not more than 15 per cent. of the cases. It must be remembered, however, that it may have disappeared before the child came under observation and had not been noticed by the mother. One of us (J. T.) has several times known a mother to deny, in good faith, that the child had ever had either a rash or snuffles, although it was noted in the case history that both had been present. The snuffling varies in loudness and character according to the extent of the local lesion. Generally it has a dry sniffing sound owing to the scantiness of the nasal secretion, but in some cases there is a copious muco-purulent and hæmorrhagic discharge, and occasionally ulceration of the mucous membrane. When this is badly affected the bridge of the nose becomes broadened and flattened, and sometimes the cartilages are destroyed and a permanent deformity of the nose results (Figs. 304 and 322).

Laryngitis, with thickening and sometimes ulceration of the mucous membrane, is common; and the hoarse cry which it causes is a most useful symptom in the diagnosis of early cases.

Fissuring of the lips (Figs. 302 to 304) *and of the anus* are very characteristic lesions. The earliest stage is an obscuration of the sharp line of demarcation between the skin and mucous membrane which is a very definite feature in the healthy infant. The fissures extend on to the skin and leave linear scars, which serve as a permanent record of their occurrence (Fig. 322). Small areas of eczema at the angles of the mouth, and condylomata there or at the anus, are occasionally seen in young infants, but are much commoner at a later stage of the disease (p. 929).

The *rash* is the commonest of all the manifestations of the disease. It was said to have been absent in only 10 per cent. of our cases. In most cases it makes its appearance between the first and seventh weeks—seldom later than the fourth month—but in rare instances it may occur for the first time much later. The rash may be present at birth. When this is so the child usually succumbs.

The most characteristic situations of the specific eruption at this age are the anus, genitals, buttocks, and thighs (Figs. 305 and 306) the eyebrows, lips and chin (Fig. 301), and the palms



FIG. 301.—Eruption in Congenital Syphilis. (Girl of 3 months.)

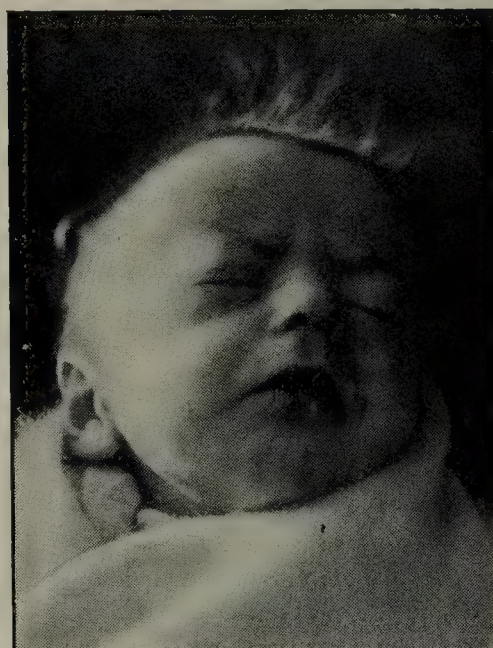


FIG. 302.—Fissured Lips in Congenital Syphilis. (Girl of 4 months.)

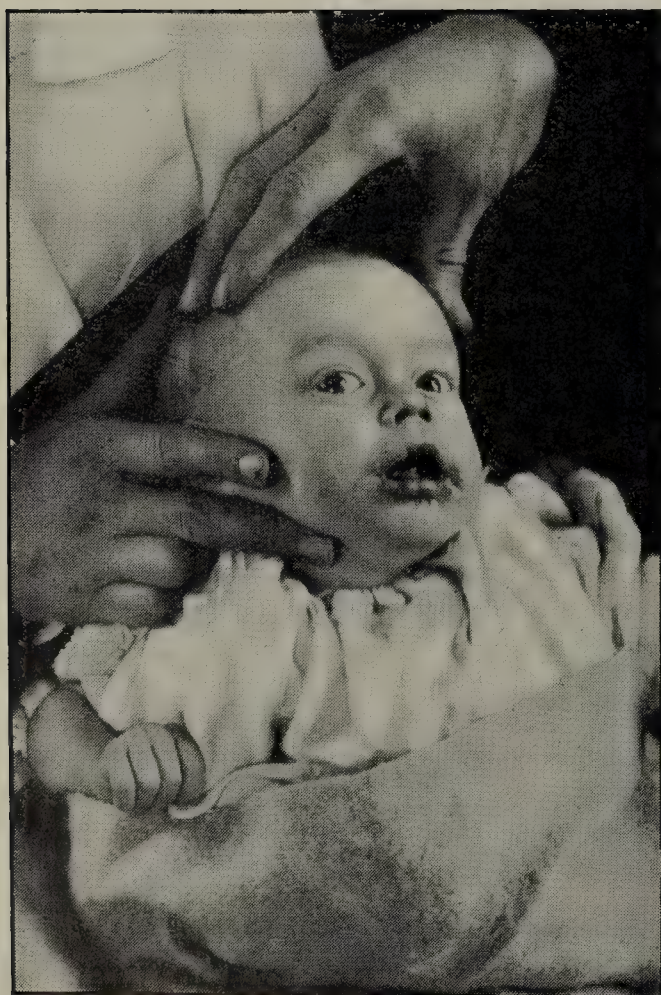


FIG. 303.—Fissured Lips in Congenital Syphilis. (Sir H. Stiles' case.)

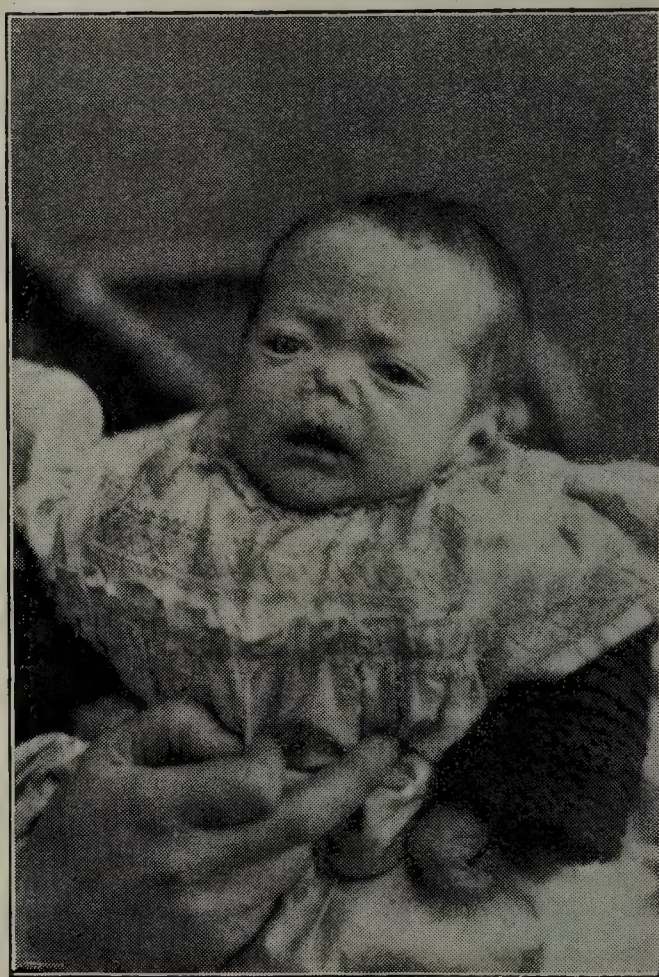


FIG. 304.—Nasal Deformity and Fissured Lips. (Boy of 3 months.)



FIG. 305.—Typical scaly macula on buttocks, legs, and soles of feet in case of Congenital Syphilis.

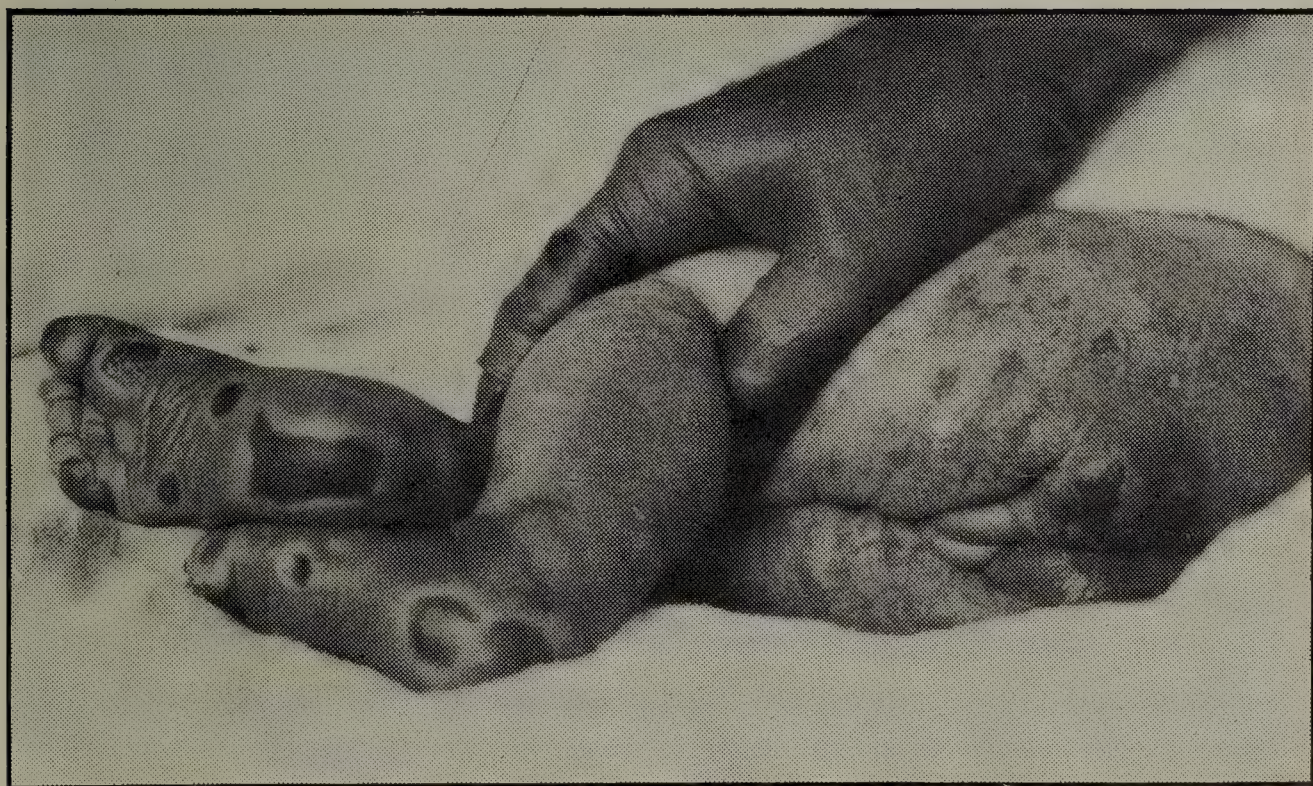


FIG. 306.—Congenital Syphilis. Pemphigus of Soles. (Dr J. S. Fowler's case.)

and soles. It varies greatly in extent and severity in different cases. In some, a few little rounded, scaly, brownish patches in one of these situations is all that is to be seen; in others, the rash may be almost universal. The front of the chest and abdomen are the places least often affected. Sometimes, when the eruption is severe, it is so complicated by eczema or erythema that its special characters are difficult to recognise.

The type of the eruption varies in different cases and in different positions. Roseola, which is so generally found in the acquired disease, scarcely ever occurs in congenital cases.



FIG. 307.—Desquamation of Soles in Congenital Syphilis, (Dr J. S. Fowler's case.)



FIG. 308.—“Syphilitic Wig.” (Girl of 10 weeks.)

The commonest lesions are small rounded scaling maculæ or large flat papules, with a yellowish-red or coppery tint, which tend to run together into blotches. Smaller papules, acuminate or flat, also occur; they also show desquamation. Pustules and boils in various situations are often met with: and, in new-born children with the disease in a severe form, bullous eruptions sometimes occur.

The palms and soles are often affected by erythema which is followed by profuse desquamation (Fig. 307); this often leaves the skin with a shiny red surface, as if it had been brushed over with gum. Occasionally purulent bullæ form in this situation in new-born children (Fig. 306).

None of the syphilitic rashes is accompanied by itching; and all of them usually yield rapidly to specific treatment.

Affections of the hair and nails are not uncommon. The scalp is often scaly and the hair scanty. In a few cases areas of alopecia appear on the forehead and elsewhere, with long hair close to them. This gives rise to the appearance which has been described by W. S. Colman and R. Hutchison as the "*syphilitic wig*" (Fig. 308). Although this appearance is most frequently seen in children with congenital syphilis, it may also occur in a typical form in other children.

In many cases the hair of the eyebrows and the eyelashes has largely disappeared. This, with the thickening of the eyelids,



FIG. 309.—Congenital Syphilis. Onychia. (Girl of 16 months.)

produces a characteristic appearance which is often helpful, in later childhood, in the diagnosis of syphilis (Fig. 330, p. 935).

Inflammation of the matrix of the nails (onychia) is occasionally met with. It may be limited to one finger or may affect them all (Fig. 309).

(c) *Disease of the Bones*.—As we have already seen (Figs. 41 to 46, p. 70), *extreme bossing of the cranium* is generally due to congenital syphilis complicated by rickets.

When *craniotabes* is extensive it also is often due to a combination of rickets and syphilis; but occasionally in young infants with untreated congenital syphilis we find a peculiar and very severe form of it without any sign of rickets.

Implication of the *long bones* is a not uncommon manifesta-

tion of congenital syphilis; in fact, Hochsinger¹ states that such will be found in practically every infant dying of the disease. Although this manifestation is classified as an early one and may be present at birth, it not infrequently makes its appearance at a later date than the other symptoms in this group.

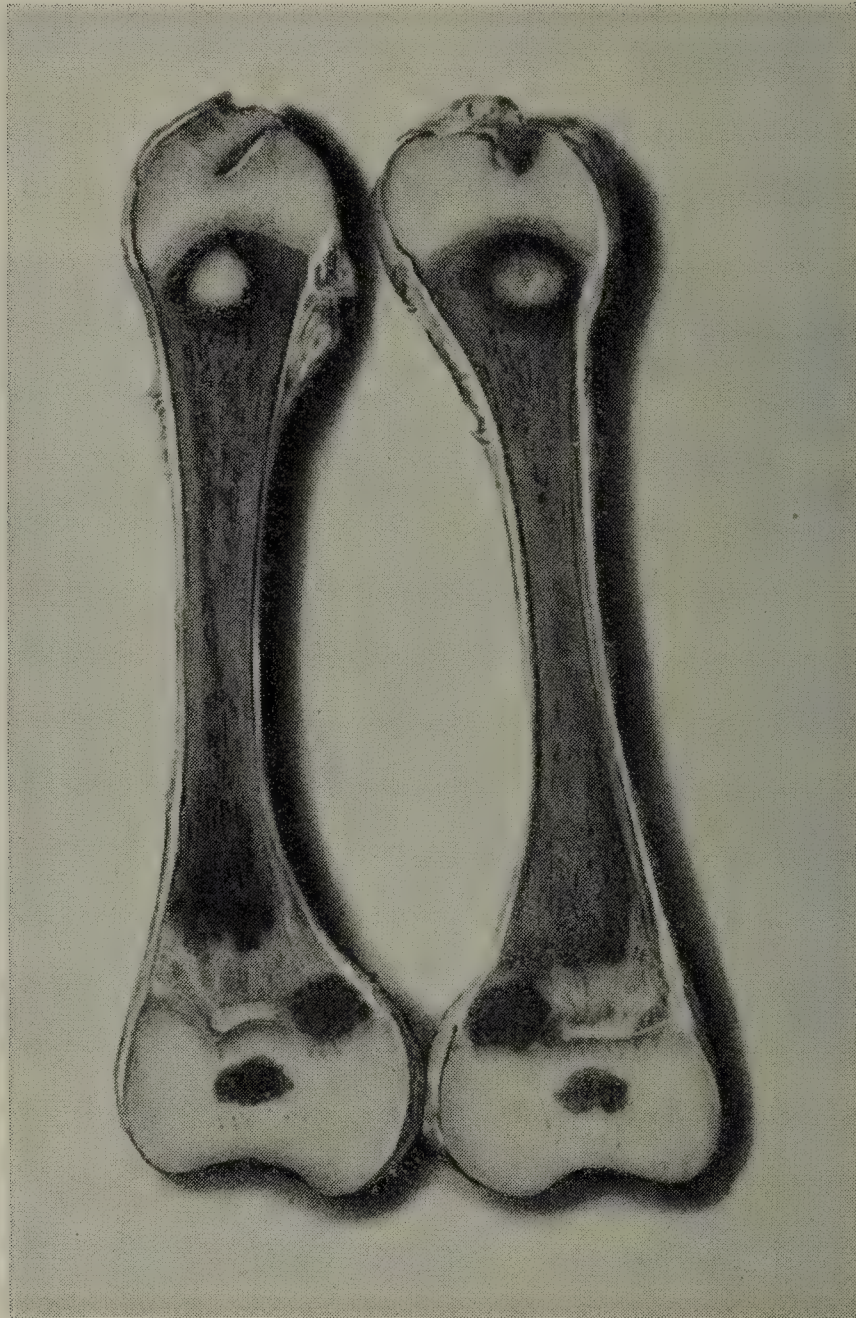


FIG. 310.—Section of bone in Syphilitic Osteochondritis, showing areas of decalcification with fatty degeneration and necrosis towards the epiphyseal ends of the diaphysis. The irregular shape and situation of the lesions explain the varied radiological appearances.

Many of the examples do not apparently develop till after the fourth month. It is often the only evident lesion.

The condition is one of osteo-chondritis and may affect the epiphyseal ends, the centre or periphery of the shaft, so that the most varied lesions result (Fig. 310). It is hence variously

¹ K. Hochsinger, *Ergeb. d. inn. Med. und Kinderh.*, 1910, v., 84.

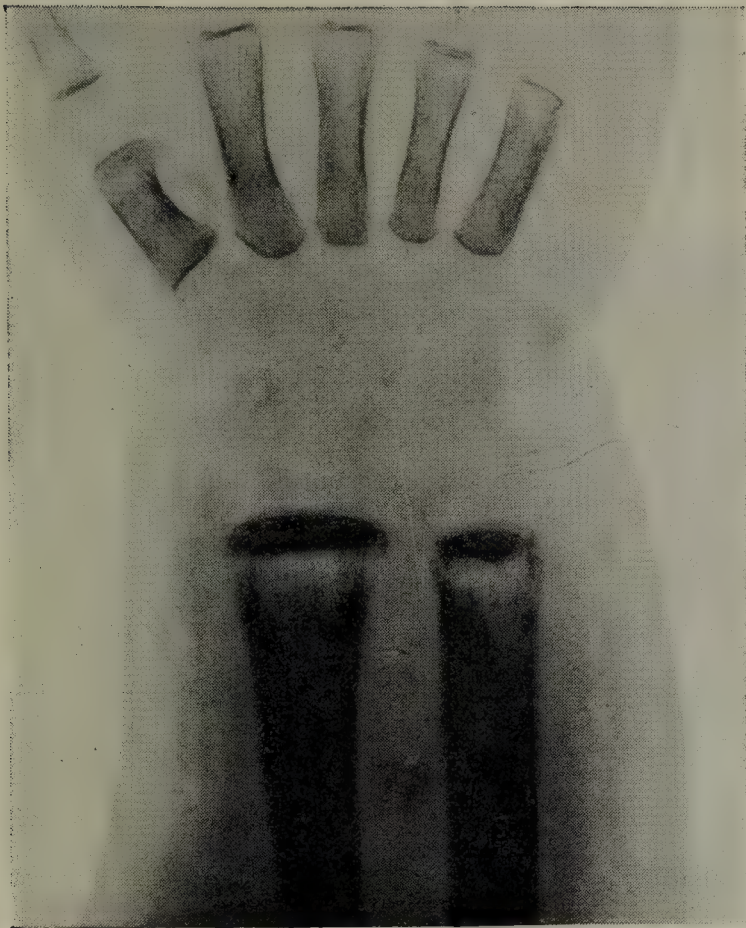


FIG. 311.—Skiagram of Syphilitic Osteochondritis with slight Periostitis. Note pallor towards epiphyseal ends of diaphysis, suggesting separation of epiphyses. Explanation of this appearance is shown in Fig. 310, p. 922.

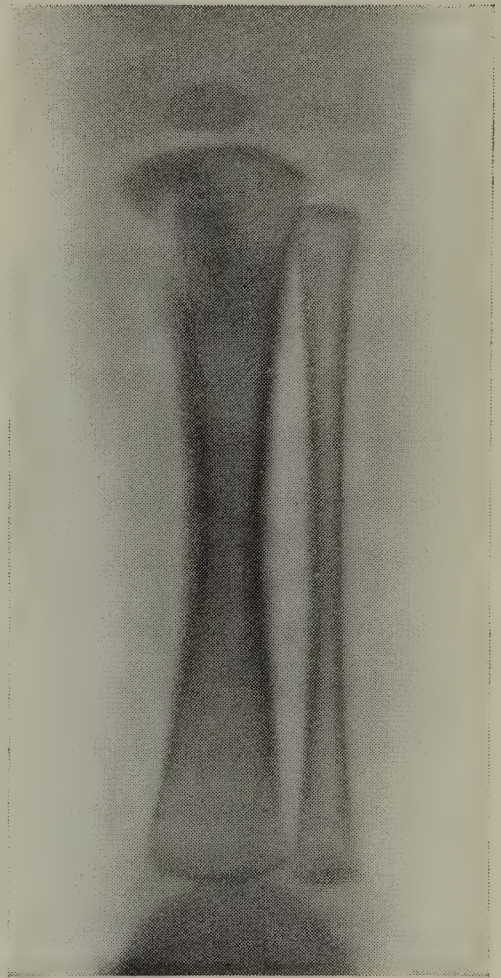


FIG. 312.—Skiagram of Syphilitic Osteochondritis and Periostitis. (Boy aged 18 weeks.)

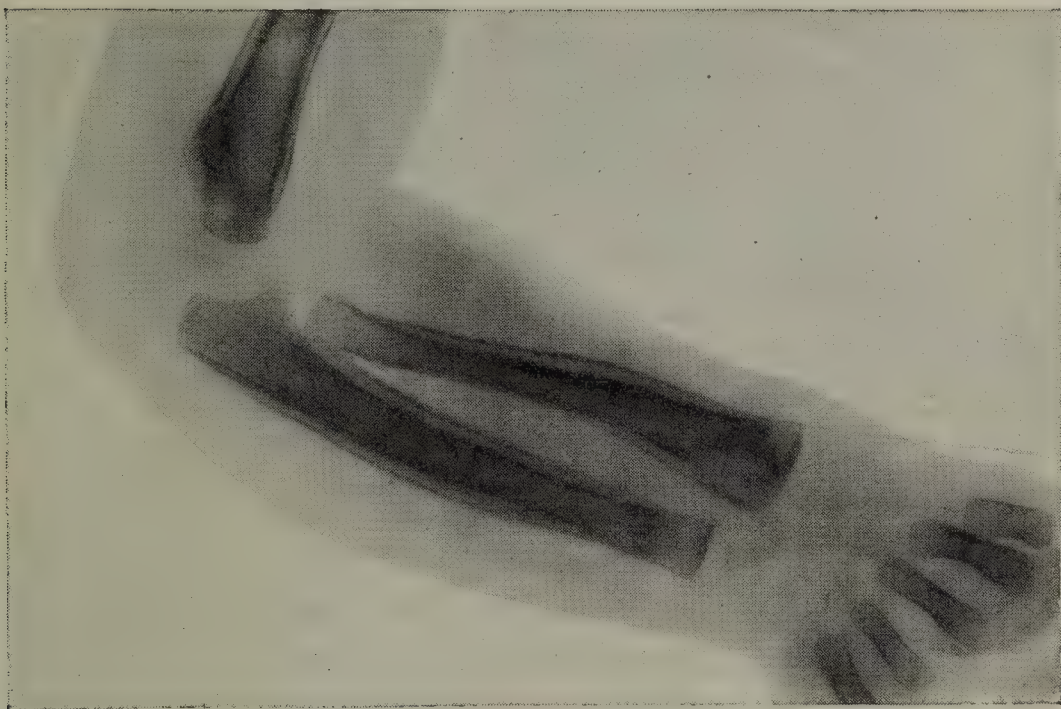


FIG. 313.—Skiagram of Syphilitic Periostitis.

called osteochondritis, epiphysitis, osteitis, and periostitis. The lesions are often, if not always, multiple and may occur in any of the limbs, but the commonest sites are the upper end of the humerus and in the vicinity of the elbows. The radius and

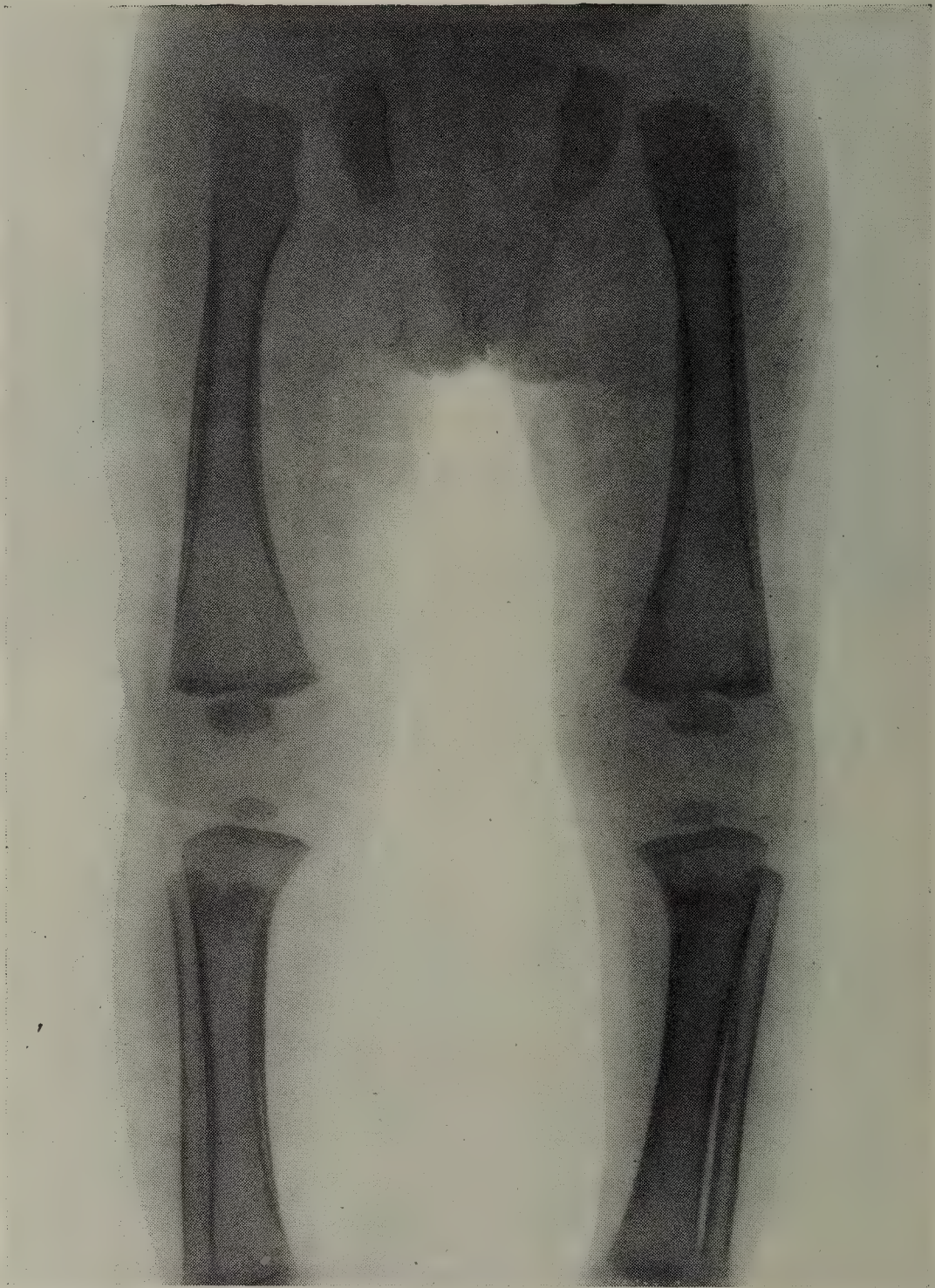


FIG. 314.—Skiagram of widespread Syphilitic Periostitis.
(Girl aged 31 weeks.)

ulna, the femur and tibia, and the metacarpus and phalanges are next in order of frequency attacked.

Attention is usually attracted to the condition by apparent pain on movement or handling, but there may be neither pain nor tenderness. There is usually, though not invariably, some swelling of the bone to be detected. A disinclination of the

infant to move the limb (Parrot's *pseudo-paralysis*) is also not uncommon. When the arm is affected the paralysis is flaccid, but it is spastic in the case of the lower limbs. The condition is best revealed by X-rays, and various are the appearances disclosed (Figs. 311 to 314).

Syphilitic dactylitis due to gummatous periostitis (Figs. 315 and 316) occurs occasionally in older infants. Joint affections are extremely rare in early infancy.



FIG. 315.—Skiagram of hand in case of Syphilitic Dactylitis, showing swelling of proximal phalanges due to periosteal new-formation. (From same case as Fig. 316.)

(d) *Visceral Lesions*.—The liver and spleen are the internal organs most frequently affected in infancy. Splenomegaly in an infant under three months of age is always to be regarded as suspicious of lues: it is found in about half the cases examined during life, and in a much larger proportion of still-born children. These enlarged spleens show no distinctive features; the condition seems to be one of simple hypertrophy. The enlargement often persists long after the other early

symptoms subside; it is apt to be specially marked in cases in which there is severe bossing of the cranial bones.

The *liver* is often severely affected, though not necessarily enlarged; and this may sometimes account for a perplexing failure of antisyphilitic treatment. In infants who die early from congenital syphilis the liver is frequently found to show interstitial hepatitis on microscopical examination, although little change may be visible to the naked eye. In some cases

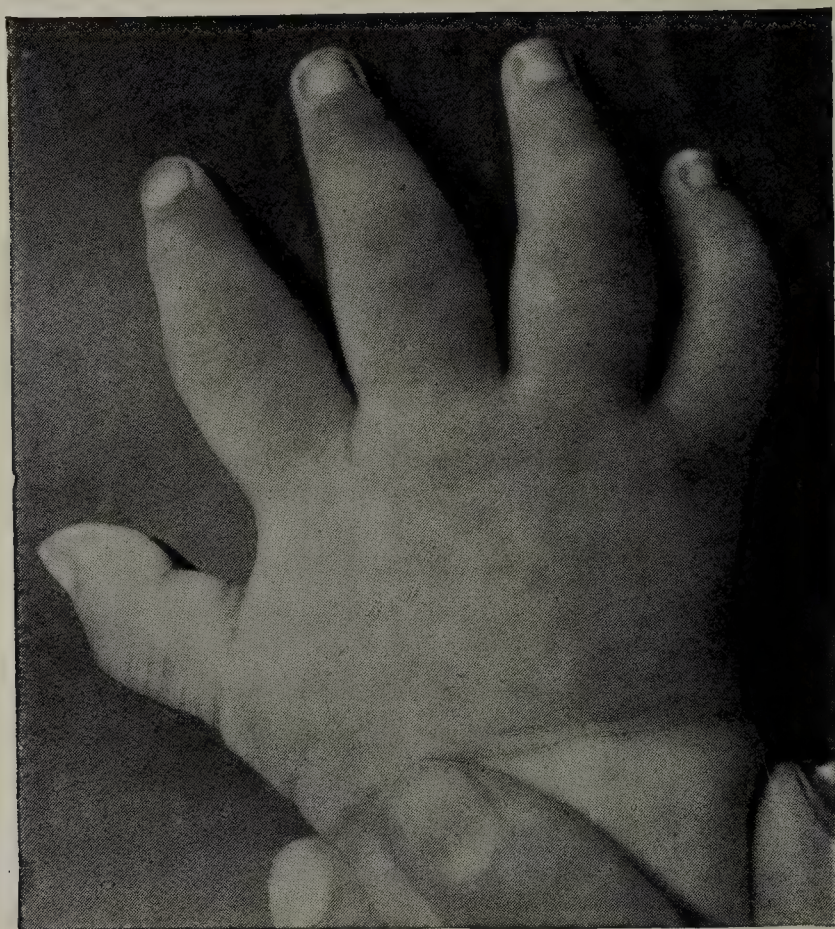


FIG. 316.—Hand in Syphilitic Dactylitis, showing flask-shaped appearance of the fingers. (From same case as Fig. 315.)

its section is studded over with miliary gummata. Acute perihepatitis, with ascites, occasionally occurs, or chronic pericholangitis with obliteration of the bile-ducts (p. 363). Large gummata do not occur at this stage. Jaundice is uncommon in syphilitic infants; when present, the case is usually fatal.

Ulceration of the *stomach and bowels* has been described but is rarely seen. The *kidneys* also are rarely affected, but a number of cases of syphilitic nephritis have been described.¹

Swelling of the *testicles*, in infants under six weeks, is generally specific in origin. It generally disappears under treatment.

¹ Geo. Carpenter, *Brit. Journ. Child. Dis.*, March 1908, v., 94.

Enlargement of the *lymphatic glands* is not so prominent a feature in congenital as in acquired syphilis; and, when present, is often due to secondary infections.

Congenital syphilis is given as one of the causes of *malformations of the heart*, but of seventeen examples observed in R.H.S.C., Glasgow, not one reacted positively to Wassermann's test.¹

Enlargement of the superficial veins on the scalp (Fig. 317) and elsewhere has been described by Edmond Fournier² and others, and is a common symptom which is sometimes useful in diagnosis, and is often a valuable aid in intravenous therapy. As has been already mentioned (p. 492), a tendency to hæmorrhage sometimes occurs³; it is probably due to disease of the blood vessels.

An interstitial affection of the *lungs*, "white pneumonia," has been described by Virchow as sometimes present at birth, or during the first few days of life.

Syphilitic affections of the nervous system are not very common, but we occasionally meet with meningitis, hydrocephalus, and encephalitis, and the latter generally leads to severe mental defect (p. 835). Many syphilitic infants take fits.

The *eyes* are not very rarely the seat of disseminated choroiditis, optic neuritis and atrophy, and irido-cyclitis. Uncomplicated iritis is rare, but occasionally occurs between the third and sixth months. Sloughing of the cornea is occasionally met with in ill-nourished babies.

In the not very common case of a syphilitic mother having a number of syphilitic children who survive early infancy, it is

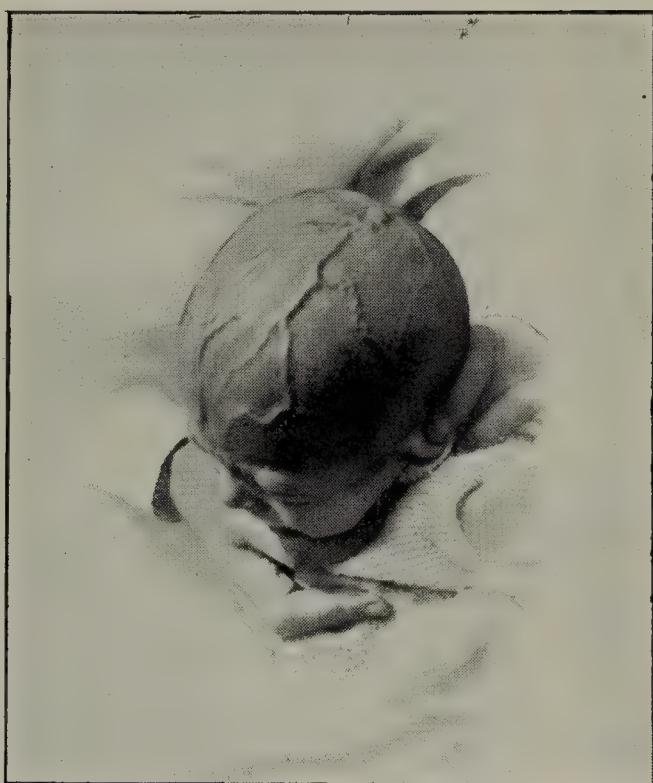


FIG. 317.—Congenital Syphilis. Enlargement of superficial veins. (Boy of 6 months.)

¹ L. Findlay, *Amer. Journ. Dis. Child.*, 1924, xxviii., 133.

² "Des Dystrophies Veineuses de l'Hérédosyphilis," *Rev. d'Hygiène et de Méd. Infant.*, 1902, i., 26.

³ Fr. Mracek, *Vierteljahrschr. f. Dermat. u. Syph.*, 1887, xiv., 117.

interesting to find that, not infrequently, one child after another shows the disease in the same organs or tissues¹—*e.g.*, in the nervous system, the bones, the liver, or the aorta.²

In dealing with syphilitic infants we have always to bear in mind that they have a strong tendency to acquire all sorts of pyogenic infections, so that they often suffer severely from non-specific as well as specific lesions. The disease also leads to a serious lowering of the general vitality, which often outlasts

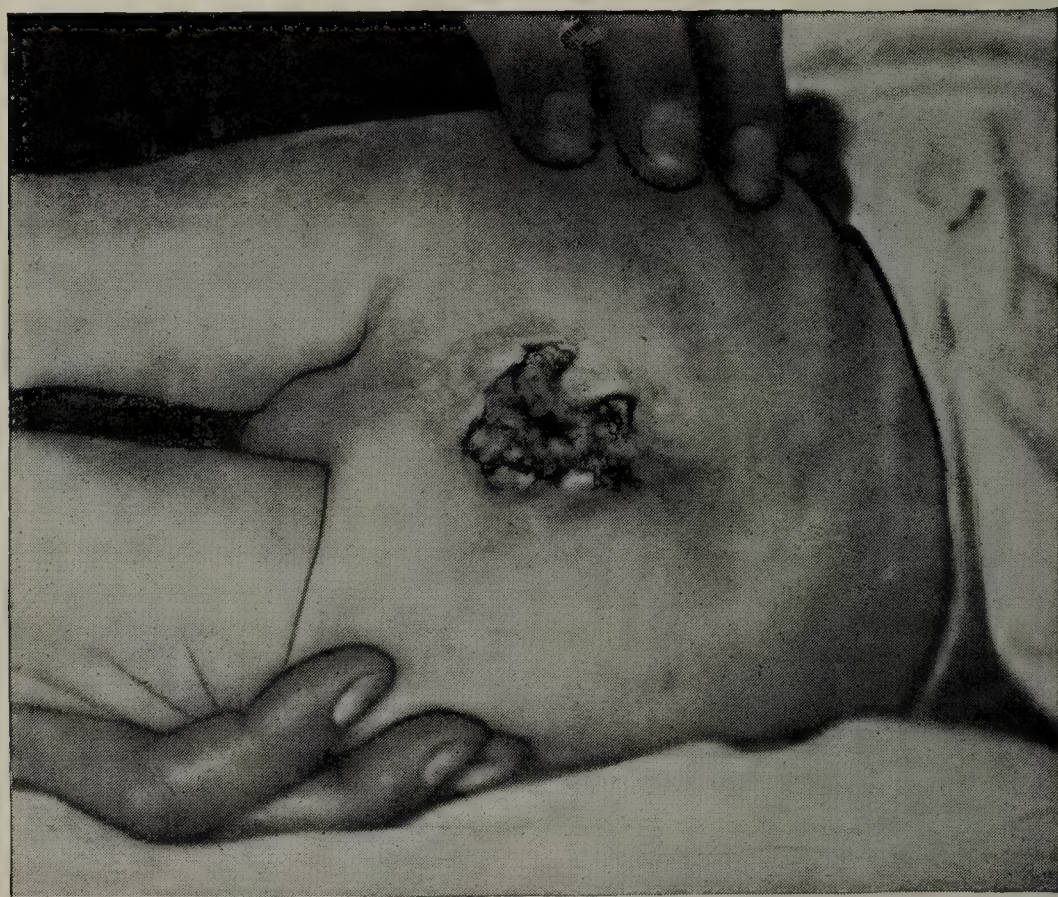


FIG. 318.—Extreme degree of Condylomatous Ulceration round Anus, of one year's duration. Note the undermining of skin at the margin of the ulceration.

its obviously specific manifestations; so that an infant who seems to have recovered entirely, and who has all the appearances of perfect health, may collapse suddenly during an acute attack of pneumonia or diarrhoea which would not have proved fatal to a healthy child. So marked is this tendency to secondary diseases that comparatively few infants, even successfully treated for the luetic infection, reach the age of one year.

2. Intermediate Symptoms.—After the early manifestations of the disease have ceased to appear, which occurs usually in a few months, or at most before the end of the first year,

¹ J. Thomson, *Scot. Med. and Surg. Journ.*, Jan. 1899, iv., 31.

² Neugebauer, *Wien. klin. Wochenschr.*, 1914, xxvii., No. 17, 503.

there are in the majority of cases no further active symptoms of a characteristic kind for three or four years or more. Occasionally, however, definite specific lesions occur, especially in the skin.

The commonest of these are *condylomata*, which are found mostly at the margin of the anus (Fig. 318), often during the second or third year. When present, they prove conclusively the syphilitic nature of the case. They are, however, more characteristic of the acquired than of the congenital form of the disease. Mucous tubercles are sometimes found at the corners of the mouth and elsewhere; and circum-oral eczema, which is commoner at a later stage, may occur.

Rarely we find other definitely specific lesions, such as syphilitic dactylitis, and gummata may form in some of the internal organs as in the liver, heart, and thyroid. We have also observed cases in which syphilitic retinitis and choroiditis occurred at this stage.

Later Manifestations.—The term “syphilis tarda” used to be applied to the manifestations of the disease which appear in later childhood—usually during the period of the second dentition—because it was noticed that, in many cases in which they occurred, no history of early symptoms could be obtained. The lesions affect many systems and organs, and resemble the tertiary manifestations of the acquired disease. Their chief varieties are as follows:—

(1) *General Symptoms.*—Before any characteristic lesions make their appearance the children are often noticed to be pale and anæmic; and they are generally stunted in growth and lacking in energy.

(2) *Affections of the Skin and Mucous Membranes.*—Skin affections are much less common in congenital than in acquired syphilis. L. Findlay and J. F. Watson¹ have, however, drawn attention to the frequency of circum-oral eczema (Fig. 319). This occurs at one or both angles of the mouth, and often spreads outwards on to the cheeks, and sometimes to the mucous membrane of the lips. The patches are red in colour and irregular in outline, and their margins may be either sharply cut or ill-defined. The surface is generally dry and scaly. The condition often occurs when there is no other definite sign of syphilis, and it is very resistant to other than

¹ *Lancet*, 1913, i., 875.

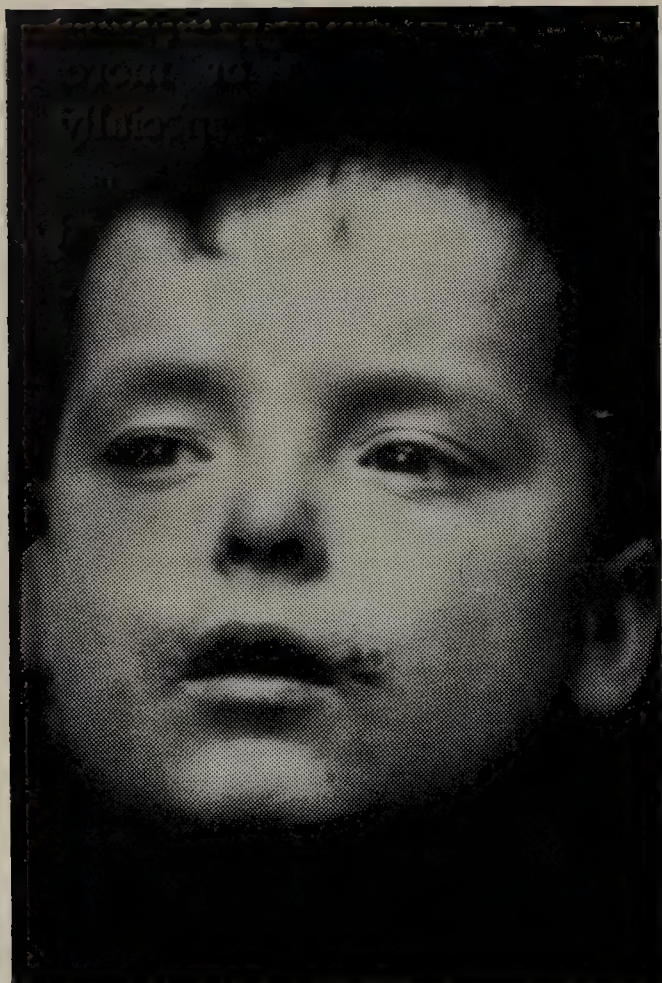


FIG. 319.—Circum-oral Eczema in Congenital Syphilis. (Boy of $4\frac{3}{4}$ years.)

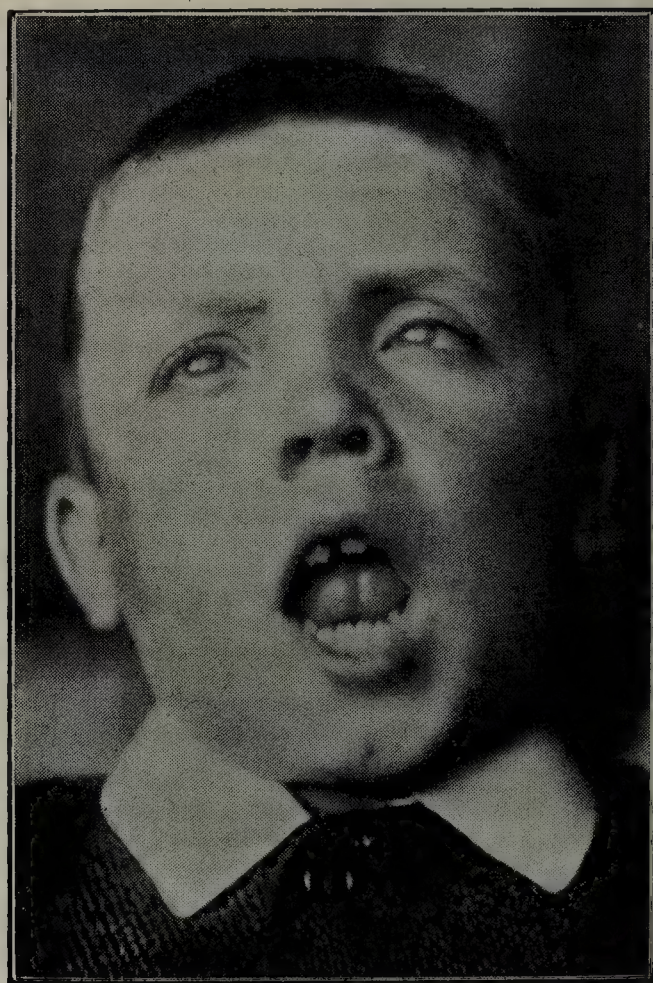


FIG. 320.—Square forehead, keratitis, depressed bridge of nose, and characteristic teeth in Congenital Syphilis. (Boy of 10 years.)

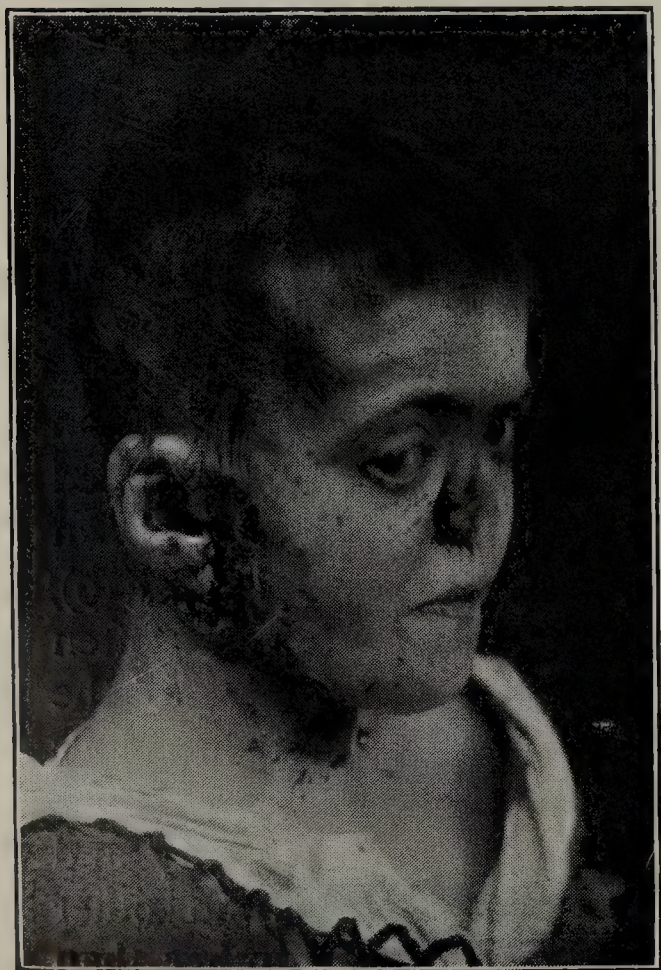


FIG. 321.—Square forehead, phagedænic ulceration of nose, ear, and neck in Congenital Syphilis. (Girl of 14 years.)

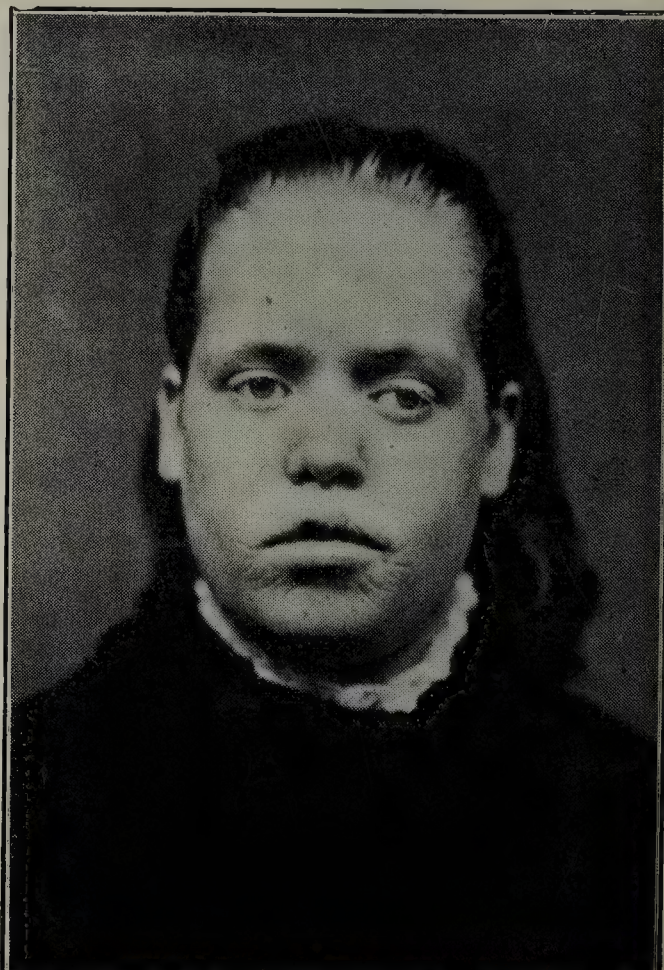


FIG. 322.—Broad bridge of nose, scarring round mouth in Congenital Syphilis. (Girl of 16 years.)

specific treatment. Its nature is only proved by the Wassermann test and by the effect of antisyphilitic treatment.

Gummatous infiltrations of the skin and mucous membranes (*e.g.*, gumma of the tongue and palate) and psoriasis palmaris are rarely met with.

Phagedænic ulceration¹ and sloughing of the skin and mucous membranes, which resemble lupus in their results, but progress much more rapidly, are not uncommon. They often affect the nose, palate, and pharynx (Fig. 321). They may also involve large areas of the skin and subcutaneous tissues on the buttocks or elsewhere on the body without any apparent local determining cause. Great loss of tissue and extensive scarring result (Fig. 323).



FIG. 323. — Congenital Syphilis. Scars from phagedænic ulceration over sacrum and left trochanter. (Boy of 9 years.)

(3) *Bone Conditions.*—

A slightly tender swelling of one or more of the long bones, with severe nocturnal pain, is one of the commonest manifestations of the disease at this stage. It is caused by specific osteo-periostitis and is commonest in the tibiæ and ulnæ, but it also may affect the femur, fibula, humerus, radius, and clavicle. It occurs in two types.

The *sclerosing type* causes much thickening of the bone with alteration in its shape and sometimes overgrowth in length. When severe, it leads to the so-called “sabre tibia” (Figs. 324 and 326). The apparent bowing of the bone in this condition is due to deposition of new bone on its anterior aspect, and not to true bending such as occurs in the case of rickety deformity of the tibiæ which bears a superficial resemblance to it (Figs. 325 and 327).

In the other variety, which is less frequently met with, there is *necrosis of the bone with gummatous softening* over it. This breaks down, leading to abscess and sinus formation. Cases of this type are apt to be mistaken for tuberculous bone disease ;

¹ J. H. Sequeira, *Lancet*, 1914, i., 11.

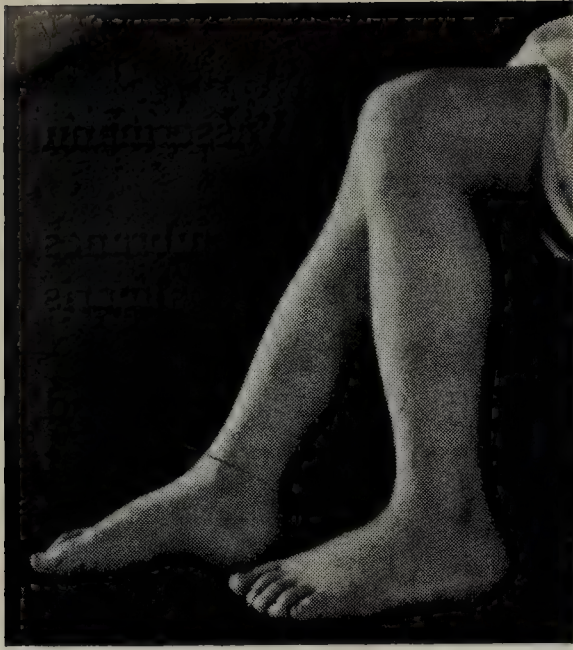


FIG. 324.—Sabre Tibia, due to Syphilitic Osteitis in a girl aged 21 years.



FIG. 325.—Tibial Curvature due to Rickets.



FIG. 326.—Skiagram of Sabre Tibia showing curving of anterior margin of bone due to deposition of new bone.



FIG. 327.—Skiagram in case of Rickets, showing that deformity is due to true bending of the bones.

but the use of X-rays, the Wassermann test, and the result of specific treatment prove their real nature (Figs. 328 and 329).

(4) *Joint Affections*.—Far the commonest syphilitic joint affection is the relatively painless, usually symmetrical arthritis, with effusion, of the knees and other joints, which has been already described (p. 82, and Fig. 52). Occasionally a painful



FIG. 328.—Skiagram showing extreme degree of Periostitis in Syphilis tarda (before treatment).



FIG. 329.—Skiagram showing absorption of new-formed bone in same case as Fig. 328 as the result of salvarsan therapy.

arthritis of one or both knee-joints with fever may simulate subacute rheumatic fever, but the absence of response to salicylates should quickly cause one to think of lues.¹ Joint mischief simulating rheumatoid arthritis² or osteo-arthritis (Still³) is also rarely observed.

¹ L. Findlay, *Rheumatic Infection in Childhood*, 1931, p. 34.

² J. W. Findlay and J. R. Riddell, *Glasgow Med. Journ.*, 1906, lxv., 13.

³ *Common Disorders and Diseases of Childhood*, 3rd edit., 1915, 812.

(5) *Visceral Disease*.—(a) The *liver* is rarely affected either with cirrhosis, amyloid disease, or gummata. (b) The *spleen* is enlarged in exceptional cases and may give rise to the picture of Banti's disease (p. 474). (c) The chief specific affection of the *kidneys* is paroxysmal hæmoglobinuria, which almost always occurs in children who are the subjects of congenital syphilis. (d) Syphilitic *endarteritis* occasionally occurs, and may lead to aortic disease. Most of the cases of aneurism in children are due to this,¹ and it is also probably responsible for the cases of hemiplegia which we sometimes meet with in syphilitic children. The subjects of Raynaud's disease also are often found to have this disease. (e) Much the most important lesion of the nervous system is that which gives rise to the form of progressive dementia, which has already been discussed in Chapter XXXII (p. 837). A few cases of *epilepsy* have been shown to be of specific origin by the presence of a positive Wassermann reaction, and by the striking effect of antisyphilitic treatment. *Gummatous tumours of the brain* are very uncommon, but a few have been reported which disappeared rapidly under treatment with mercury and iodide. (f) *Affections of the eyes*.—Interstitial keratitis is one of the commonest late manifestations of the disease. It usually begins insidiously with a slight cloudiness of the cornea, without pain (Fig. 320). This, after gradually increasing for a time, slowly disappears, and in most cases leaves the sight unaffected. In severe cases, however, a certain amount of permanent haziness, and occasionally dense leucomatous patches, are left which interfere with vision. The condition is curious in this respect, that, although certainly due to congenital syphilis in the majority of cases, it differs from nearly all the other manifestations of the disease in being comparatively little influenced by specific treatment. We have several times seen it develop in children who had been under constant treatment with mercury and iodide for weeks or months, and in one case it began after the Wassermann reaction had become negative as the result of a prolonged course of neo-salvarsan and mercury. *Iritis* may occur alone, or along with keratitis, and choroiditis also often develops at this stage. (g) The disease of the ears, which is characteristic of congenital syphilis, consists in an *affection of the internal ear* which rapidly

¹ E. Bronson and G. A. Sutherland, "Ruptured Aneurisms in Childhood," *Brit. Journ. Child. Dis.*, 1918, xv., 241.

leads, in most cases, to complete and permanent deafness. It generally occurs after puberty, and is often bilateral. (*h*) The peculiar dental deformity known as "Hutchinson's teeth," though only seen at this period of life, is due to disease in early infancy. It has been considered in Chapter III (p. 63).

Diagnosis.—It is rare to get much help in the diagnosis of congenital syphilis from interrogating the mother about her own symptoms. We have generally to trust to the history, and to the presence of the early manifestations of the disease which we have described. If the Wassermann reaction can be done, either in the child or in the mother, by a competent bacteriologist, it is generally conclusive at all ages. We frequently obtain no history of specific symptoms in the mother.

In the *intermediate* stage of the disease our suspicions should always be aroused if we find thickening of the eyelids, with loss of eyelashes or signs of past iritis, especially if there

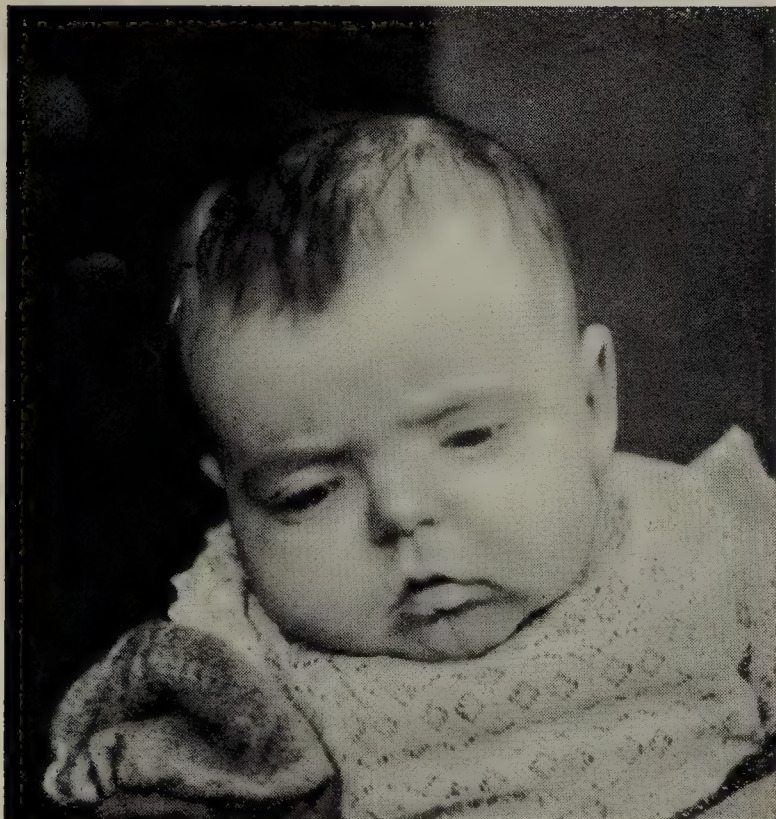


FIG. 330.—Congenital Syphilis. Loss of eyelashes, thickened eyelids, atrophy of alæ nasi. (Same case as Fig. 302, at 12 months.)

is also obstruction of the nasal passages with atrophy of the alæ nasi (Fig. 330). Fissuring of the mucous membrane of the lips and adjoining skin, and a severe degree of depression of the bridge of the nose, are almost sure indications of the presence of the disease (Fig. 322). Prominent bossing or thickening of the cranial bones also suggests strongly the presence of syphilis; and, when this condition is met with along with evidence of a moderate amount of past hydrocephalus, it may be considered pathognomonic.

In *later childhood and adolescence* the presence of a congenital syphilitic taint may be diagnosed (*a*) if traces of past syphilitic disease in infancy are found; (*b*) if the morbid condition present is of an undoubtedly specific character.

(a) *Traces of past Syphilitic Disease.*—Many syphilitic children grow up showing no trace of their past specific ailments. The permanent alterations of the teeth, bones, etc., are only found in a minority of the cases, so that their absence proves nothing. The traces of past syphilitic disease which are most often found are the following :—

(1) The characteristic deformity of the permanent teeth, the importance of which has already been mentioned (p. 63).

(2) Absorption patches in the choroid, usually situated towards the periphery.

(3) Bossing of the head in older children, although often less distinct than that seen in young infants, is important. It is usually represented in older children by a general thickening of the bone which gives the forehead a heavy, square appearance.

(4) Depression or broadening of the bridge of the nose.

(5) Fissuring of the lips and adjacent skin.

(b) *Essentially or probably Specific Lesions.*—The commonest of the morbid conditions which are so characteristic of congenital syphilis that their presence is suspicious, or even pathognomonic of the disease, are as follows :—

(1) Interstitial keratitis.

(2) Sudden incurable deafness from disease of the internal ears.

(3) The occurrence—most commonly on the tibiæ—of periosteal nodes which are painful at night.

(4) The spontaneous occurrence of subacute or chronic synovitis of both knees, with comparatively little pain or stiffness. This is common in syphilis, and very rare from any other cause.

(5) Phagedænic ulceration of one of the mucous membranes such as that of the nose, palate, or throat, or of the skin.

(6) Circum-oral eczema.

(7) Progressive dementia.

Treatment.

There can be little doubt that in the past both the prophylaxis and the curative treatment of congenital syphilis have been most unsatisfactory. The former has not always been carried out on a wide enough scale; and the latter has too often been applied to the child alone; and, even in his case, has

sometimes aimed too exclusively at relieving the present symptoms, and too little at freeing him completely and permanently from the disease.

On undertaking a case of congenital syphilis, we must remember that we are dealing with *a family disease*. We must also realise that, provided we are allowed to use the means now at our disposal, we may fairly expect, in most cases, to banish it for good and all from the whole family, as well as from the patient.

Our treatment of the child therefore forms only a part of our duty, for that of the family is at least as important. With this in view, the Wassermann reactions of the father and mother must always be taken, if they will allow it; and, if they are positive, they must be transferred, without delay, to clinics where they will be thoroughly treated and kept under constant observation until they have completely recovered. The other children also must be similarly tested; and, if positive, they too must be subjected to treatment, and, in any case, to prolonged supervision.

It may, indeed, be some years before the whole family is restored to health, and there are unfortunately parents whom no amount of tact will induce to submit to the necessary examinations and to prolonged therapeutic measures. Still, thorough cure of all the affected members of the family, and nothing short of it, should always be our aim.

Prophylaxis.—The measures to be taken with a view to the prevention of congenital syphilis are as follows:—

(1) All cases of acquired syphilis must be treated as early and as thoroughly as possible; and, to this end, notification of the disease is most desirable. While there are, of course, obvious reasons why it will be very difficult to bring this into operation, there can be no doubt that it should be introduced, and that this will be done in time.

(2) The marriage of a syphilitic person should be delayed until he has been four years under treatment, and has had no symptoms of the disease for two years at least. If this precaution is carried out, it will be sufficient in such a large proportion of the cases that the risk of exceptional failure may be taken.

If the patients have been subjected to early and thorough treatment with salvarsan combined with mercury until the Wassermann reaction has become negative and has remained

constantly so for a year, marriage may be safely allowed. If the treatment has only begun at a late period of the disease, the risk is far greater, as the reaction is apt, in these circumstances, to become positive again.

(3) When a syphilitic mother becomes pregnant she must always be subjected to thorough treatment with mercurial inunction and salvarsan. If this is done there is every chance that the child will not only be born healthy, but will remain so; and, even if the treatment is intermitted after delivery, the subsequent children will also most probably be free from the disease. It may be that owing to the increased vascularity of the uterine tissues during pregnancy, or perhaps on account of some subtle change occurring at this time, the treatment is rendered more effective. The injections may be followed by headache and sickness for twelve hours, but the patients feel well at other times, and there is no interference with the course of gestation. When the syphilitic mother is properly treated the health of the child can almost always be guaranteed.

Curative Treatment.¹—The three drugs which are most useful in the treatment of congenital syphilis are mercury, arsenic, and bismuth; and, as a rule, all three should be used. While mercury still retains its place as chief among the three, bismuth is steadily gaining a reputation which bids fair to surpass that of mercury, and arsenic is a valuable adjuvant of both. Iodine, in the form of the French tincture of iodine, or *syrupus ferri iodidi*, is also very useful in the later manifestations of the disease, and especially in bone conditions.

Mercury.—Grey powder, in doses of $\frac{1}{2}$ to 1 gr. thrice daily and 2 gr. in older children, has been the form of mercury most commonly used in this country, and, if there is a tendency to diarrhoea, small doses of Dover's powder may be added; 10 to 30 minims of the solution of the perchloride (B.P.) may also be given. The *most satisfactory way*, however, of using the drug is by *inunction*. This, if it is properly carried out, is not only more effectual than grey powder, but has the advantage that it does not set up diarrhoea. A piece of unguentum hydrargyri, the size of a pea, should be used once a day—small fragments of it being rubbed into the skin until they disappear. On the

¹ This section was originally supplied by Dr Mary Macnicol, Medical Officer in charge of the V.D. Department, Edinburgh Hospital for Women and Children, Bruntsfield.

first day the inunction should be made into the abdomen, and on the second into the back; then one day may be given to each axilla and to each groin, and then the abdomen used again, and so on. The skin must be washed with soap and water before each application. The occurrence of mercurial stomatitis in infancy is extremely rare.

During the second year the inunctions should be repeated regularly every two months, and the Wassermann reaction taken from time to time. If condylomata or mucous papules are present, they should be dusted with calomel.

At all ages careful tonic as well as mercurial treatment is of great importance in the later stages; and cod-liver oil, iodide of iron, and other chalybeate preparations are often useful. Dietetic and hygienic details must also be attended to.

Arsenic.—This drug is practically never administered now in the form of *salvarsan* (606) on account of its toxicity, and the consequent likelihood of producing severe reactions, both local and otherwise. The various forms of *neo-salvarsan* (914) are almost invariably used now, both for adults and children. The commonest and most conveniently used of these preparations are novarsenobillon (N.A.B.), neo-kharsivan, sulfarsenol, and galyl.

1. *Novarsenobillon*.—This preparation is very reliable and efficacious, and may be administered either intravenously, dissolved in distilled water, or intramuscularly, in a guaiacol and glucose solution.

Dosage.—(a) Infants up to two years of age. Eight weekly injections, beginning with 0.05 gramme, and rising gradually to 0.1 gramme, should be given. The total amount administered should be about 0.6 gramme.

(b) Children between two and twelve years. A similar course should be given, beginning with 0.05 gramme, and rising more rapidly to 0.2 gramme.

2. *Neo-kharsivan*.—This is administered in similar doses and solutions to novarsenobillon.

3. *Sulfarsenol*.—This preparation may be given dissolved in distilled water, either intravenously or intramuscularly. It gives rise to no pain or other symptom of irritation and is easily borne by children. Its dosage is similar to that of N.A.B.

4. *Galyl*.—This is obtained in ampoules containing 0.4 gramme, together with a glucose base for dissolving the drug.

Very good results have been obtained from the use of this preparation, which seems to be tolerated well by very young infants. It is administered intramuscularly in doses of 0.05 gramme to 0.1 gramme.

Method of Administration.—1. *Intramuscular Injection.*—In the case of infants or young children it is very much easier and safer to administer arsenic in this way than by intravenous injection. Provided that a fine, sharp needle is used, the operation causes the child very little discomfort; and the intramuscular route has this further advantage that the drug is eliminated more slowly from the body. In the case of infants the gluteal region is the most suitable site for injection; but in the case of older children it is found that injections into the muscles of the upper arm are less alarming to the child.

The skin should be cleansed with ether or iodine; the needle, separate from the syringe, inserted quickly and deeply; the syringe with the solution connected; and the injection made steadily and slowly. The needle is then quickly withdrawn, and the part gently rubbed with a small pad of cotton wool. No local reaction should follow an injection of this kind if every precaution has been taken to render the syringe, needle, and skin aseptic.

2. *Intravenous Injection.*—Where it is decided to employ this method, the most convenient vein, in the case of infants, is found on the scalp, a branch of the temporal vein. The longitudinal sinus is also a convenient site, but it has the disadvantage that if any escapes outside the vessel the cerebrum is irritated. In older children the median basilic vein at the bend of the elbow is the position of choice.

It is important not to use too sharp or fine a needle; a No. 1 sharpened at an angle of 45° is very suitable. Great care must be taken that none of the solution enters the tissues around the vein, as acute inflammation and possibly necrosis may ensue. This method of administration has to a great extent been superseded by that of intramuscular injection, in the case of infants and young children, owing to the obvious advantages possessed by the latter method.

Bismuth.—This metal is being more and more employed in the treatment of all forms of syphilis, and very good results have been obtained in congenital cases from its use. It would appear that of all the various forms of bismuth, metallic bismuth,

finely subdivided, is the most potent in its parasiticial properties. This may be obtained for purposes of treatment in various forms, two of the commonest being *bicreol* (a cream made up of metallic bismuth and camphor) and *bismostab*, a suspension of bismuth in a 5 per cent. glucose solution.

1. *Bicreol* should be given at weekly intervals commencing with 0.5 c.c., and rising gradually to 1 c.c. in the case of infants, and 2 c.c. in the case of older children. Twelve injections should suffice for the course.

2. *Bismostab* is sold in ampoules containing 5 c.c. A course of twelve injections, beginning with 0.5 c.c., and rising gradually to 1.5 c.c., or 2 c.c., is suitable for children.

Method of Administration.—Both the above preparations of bismuth are given by intramuscular injection, the site of election being the gluteal muscles. Care must be taken that the point of the needle does not enter the lumen of a vessel, as there is danger in injecting metallic bismuth into the blood-stream. The needle should be inserted separated from the syringe; and, if blood appears at its base, it must be removed and inserted in a slightly different position. It is good practice to detach the syringe after making the injection, with the needle still *in situ*, and to inject about 1 c.c. of air to clear the needle of bismuth. This prevents the leaving of a track of bismuth along the path of the needle, and avoids consequent pain. After the injection the area round the site should be gently massaged for a few minutes.

Summary.—The best results would seem to be obtained by a judicious combination of all the three drugs mentioned above—mercury, arsenic, and bismuth.

The following is an example of a course of treatment which has given eminently satisfactory results in many cases:—

1. A course of mercurial inunction or grey powder for three or four months, combined with an eight weeks' course of intramuscular injections of galyl, sulfarsenol or neo-kharsivan.

2. Then, extending over two months, a course of *bicreol* or *bismostab*, combined with liquor arsenicalis by the mouth.

3. And, finally, a further course of mercurial inunction or grey powder for three or four months, followed by a rest, during which syrupus ferri iodidi may be given with advantage.

These various methods of treatment should be carried out in succession

An infant treated systematically and thoroughly in this way will almost certainly become free from all syphilitic manifestations, and will also probably give a negative Wassermann reaction. If the Wassermann reaction remains positive, the whole course may be repeated during the second year. The best results are naturally obtained in cases where treatment has been begun early.

Acquired Syphilis.

Acquired syphilis is occasionally seen in infancy and childhood, and its symptoms and treatment differ in no important respect from those of the same disease in adults.

The primary sore is most frequently found in the mouth or on the lips. This may be due to the infection having been communicated directly by kissing, or probably quite as often from the child's indiscriminate habit of picking up all sorts of dirty things and putting them into his mouth.

The symptoms, apart from the absence of snuffles, are similar to those of the hereditary form of the disease. Condylomata are a prominent feature (Fig. 318, p. 928). The acquired disease, as has been already mentioned, is very apt to spread by contagion.

CHAPTER XXXVII

TUBERCULOSIS

ALTHOUGH tuberculosis is fortunately diminishing, it still remains one of the commonest as well as one of the most serious diseases of childhood. It is a disease, too, which should have for us an added importance, since we are in possession of knowledge which, if applied, could bring about a still further and noteworthy diminution in its frequency.

Active and Latent Tuberculosis.—In considering the question of tuberculosis it must be borne in mind that we have to deal with two types of the disease. In the one—*active tuberculosis*—the disease has produced lesions which are causing local or general symptoms, interfering actively with the child's health and threatening his life. The other type—*latent tuberculosis*—is present in the body in such forms and positions that it may not give rise to any lesions demonstrable by ordinary clinical methods, and does not apparently interfere with the child's well-being, or prevent his gaining in weight and vigour. This condition, which we may also call *tuberculous infection*, is very much commoner than active tuberculosis. Although not suffering from the effect of the disease, these patients are nevertheless tuberculous children. Under favourable conditions the latent tuberculous infection which they harbour may, and very likely will, remain harmless during a long lifetime. It continues, however, to be a menace to the child's health, since any transient local irritation or lowering of the general resistance may arouse it dangerously from its state of quiescence.

From a pathological point of view it might seem superfluous to dwell so much on the differences between these two groups of tuberculous cases. Clinically, however, it is most important to do so. Their confusion has so often aggravated the difficulties of interpreting the clinical and pathological facts, as well as the proper line of treatment to be adopted, that we should always try to recognise, if possible, to which type any case that we are called to treat belongs.

Frequency of Tuberculosis in Childhood.—It is not possible to express with any degree of precision the incidence of the

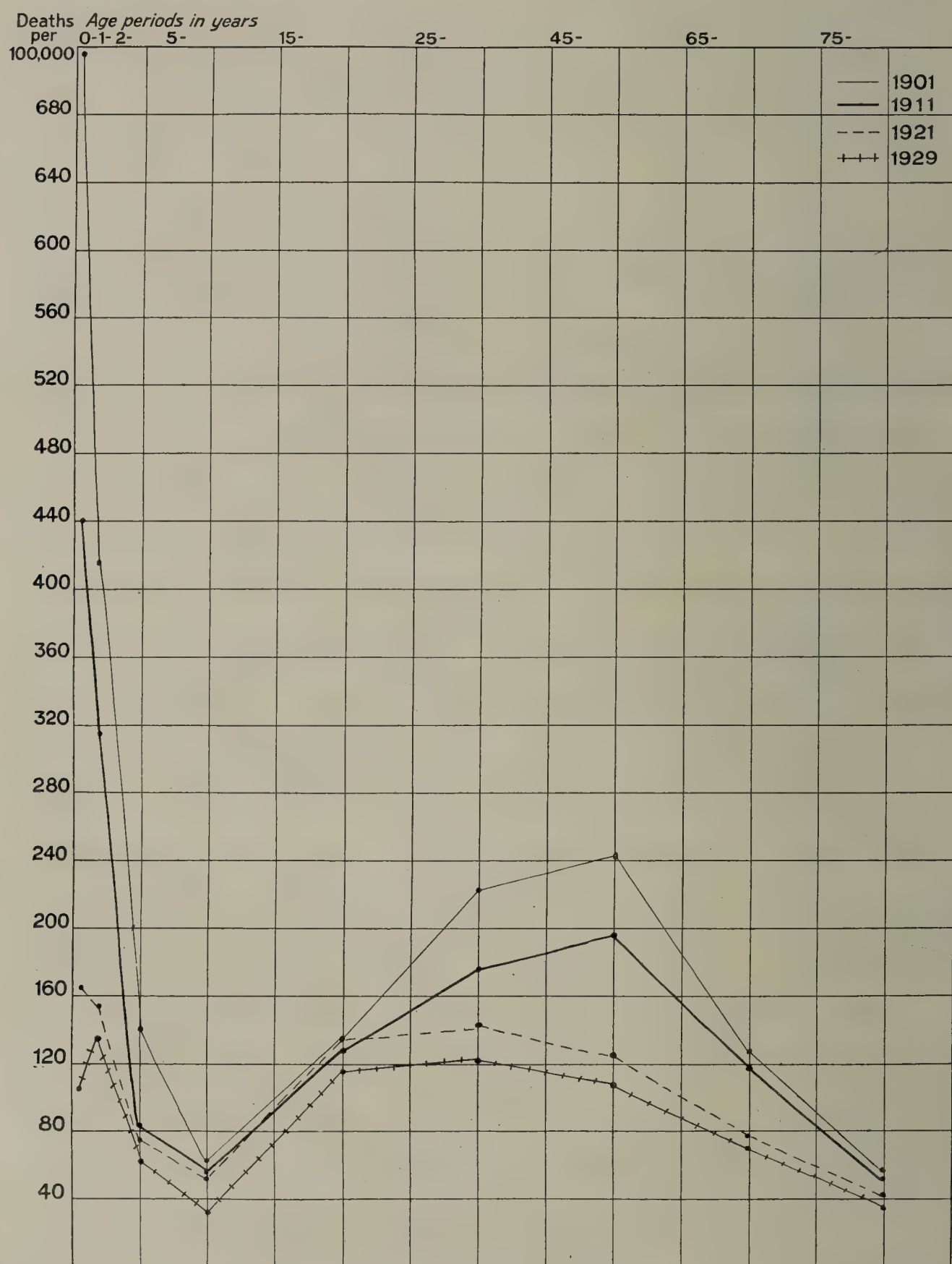


FIG. 331.—Death-Rates per 100,000 population from Tuberculosis (all forms) for different Age-Periods for various years from 1901 to 1929 (England and Wales).

tuberculous infection in childhood, for the simple reason that there is not available any extensive and accurate survey of the general child population. Although the disease is notifiable it

is only so in the active stage, and hence the returns from Public Health Authorities ignore entirely the larger group of latent mischief.

Our earliest estimates of the extent of the disease were obtained from mortality returns. These refer, of course, only to the fatal cases, and leave out of consideration the mischief which has healed, apart altogether from that large group of cases, viz., the latent varieties of the disease. These figures do show, however, that there has been during the last fifty years a steady decline in mortality from all varieties of tuberculosis (Fig. 331).

With the introduction of notification a truer index of the incidence of the disease was obtained, since all active forms are recorded. It is even possible that these figures for some varieties, *e.g.*, abdominal tuberculosis, exaggerate the amount of the mischief, because all sorts of indefinite illness with wasting are notified as such. On the whole, however, notification returns still underestimate the extent of the disease, because they also do not embrace the latent forms of tuberculosis.

The first attempts to gain information on this aspect of the question were made in the post-mortem room. In the following Table is shown the frequency with which a tuberculous focus has

Incidence of Tuberculosis found Post-mortem in Children.

Place.	Author.	Date.	Number of P. Ms.	Age of Subject in Years.	Per cent. Incidence.	Per cent. of Lesions Active.
London .	Ancell ¹ . .	1853	154	0 to 15	28.0	...
Munich .	Müller ² . .	1889	500	0 „ 15	42.0	...
Zürich .	Naegeli ³ . .	1900	88	0 „ 17	16.0	69
Berlin .	Baginsky ⁴ . .	1903	5448	?	26.9	...
Dresden.	Burckhardt ⁵ . .	1903	190	0 to 15	40.0	50
Kiel .	Heller ⁶ . .	1903	...	$1\frac{3}{2}$ „ 15	20.1	...
Berlin .	Orth ⁷ . .	1904	418	0 „ 15	11.0	...
Vienna .	Hamburger and Schluka ⁸ . .	1905	401	0 „ 14	40.0	68
Vienna .	Albrecht ⁹ . .	1909	3213	0 „ 12	33.0	...
Glasgow .	Blacklock ¹⁰ . .	1932	1800	0 „ 13	15.7	90

¹ *Assoc. Med. Journ.*, 1853, p. 1038.

² *Münch. Med. Woch.*, 1889, Nos. 50 and 51.

³ *Virch. Arch.*, 1900, clx., 426.

⁴ *Berl. Klin. Woch.*, 1903, xl., 223.

⁵ *Münch. Med. Woch.*, 1903, l., 1275.

⁶ *Diss. Kiel*, 1903.

⁷ *Berl. Klin. Woch.*, 1904, 265.

⁸ *Jahrb. f. Kinderh.*, 1905, lxii., 517.

⁹ *Wien. Klin. Woch.*, 1909, xxii., 327.

¹⁰ *Proc. Roy. Soc. Med.*, 1932, xxv. (Path. Sect., p. 11).

been found during the course of autopsies in children in different centres. In the majority of instances no discrimination is made between active and latent mischief, although a few authors do state in what proportion of the tubercular cases the disease was the cause of death, and in these instances this would seem to be between one-half and two-thirds of the total.

These findings show that the incidence of the disease is considerable and that it varies in different communities, but as they only refer to the hospital class, and the sick of this class, one must be careful not to draw far-reaching conclusions.

It is, however, only from the results of *tuberculin tests* that we can obtain the truth concerning the real incidence of infection by the tubercle bacillus during childhood. This test can be applied in several ways. The original method of the injection of a definite amount of tuberculin hypodermically is not very generally employed to-day. It is a method which has certain drawbacks. It is a quantitative test, *i.e.*, a positive reaction is obtained even in the non-tuberculous if the dose is large enough, and hence careful graduation of the amount of a standardised tuberculin is required. It is recommended that a dose not exceeding $\frac{3}{10}$ mgrm. be commenced with and gradually increased to 10 mgrm. if a reaction is not obtained with the smaller dose. The reaction is both local and general. The former is specific and consists of a congestion of the tuberculous focus wherever situated. If this lesion is in the lung, crepitations may appear. The general reaction is of the nature of a feeling of malaise and a rise in the temperature, which, to be significant, must reach at least 100° F. from four to thirty-six hours after the injection. This makes the test unsuitable for patients with fever. It is thus seen that there are many factors to be controlled, and it is for this reason that it is not a popular test. Nevertheless, it is considered by some workers to be still the best test for tuberculosis, and, if intelligently applied, to be without danger.

In 1907, Pirquet¹ introduced the *skin test* and Calmette² the *ophthalmic test*. The latter consists in the instilling into the conjunctiva of a dilute solution (1 per cent.) of tuberculin, which causes in the tuberculous individual a conjunctivitis. Since, however, a condition analogous to strumous ophthalmia has occasionally developed, the method is practically never

¹ C. von Pirquet, *Deut. Med. Woch.*, 1907, xxxiii., 905.

² A. Calmette, *Compt. rend. Acad. d. Sc.*, 1907, cxliv., 1324.

employed. Pirquet's test is performed by scarifying the skin through a drop of Koch's old tuberculin, much in the same way as in ordinary vaccination. This method, in contrast to the subcutaneous technique, may be considered truly specific, as sufficient toxin is never absorbed to cause a reaction in the non-tuberculous subject. When positive, there develops at the seat of scarification, twenty-four or more hours later, an area of hyperæmia, or even œdema, and in some instances vesiculation with necrosis and ulceration may result. It is customary when performing Pirquet's test to control the result by scarifying the skin in the same way at another place through an aqueous solution of glycerine.

Moro¹ some time afterwards showed that if an ointment containing tuberculin were simply rubbed into the skin, folliculitis in the tuberculous child resulted.

The great objection to Pirquet's and Moro's tests is that the amount of the tuberculin introduced into the skin cannot be controlled, and hence the degree of the reaction, or even its mere presence, is somewhat erratic. In order to overcome this difficulty Mantoux² in 1908 introduced the method of the *intradermic injection* of a definite amount of the drug (0.1 c.c. of 1/1000 solution of Koch's old tuberculin, equal to $\frac{1}{10}$ mgrm.). If no reaction results with this dose then 0.1 c.c. of 1/500 solution ($\frac{1}{2}$ mgrm.), and if the result is still negative, 0.1 c.c. of 1/100 solution (1.0 mgrm.) should be used. The reaction consists in the development, twenty-four or forty-eight hours later, of an area of erythema with a central zone of œdema, which latter should measure in diameter at least 1.5 cm. At times the reaction is delayed for five or six days. This reaction is specific for tuberculosis, but it should be remembered that, like all tuberculin reactions, it does not differentiate between the human and bovine types of infection.

It is nowadays generally recognised that Mantoux's test gives the largest proportion of positive results, and it is from the findings with this test that the best index of the incidence of tuberculosis is obtained. Blacklock found that, of 157 Pirquet reactors coming to post-mortem examination, 20 (12.7 per cent.), whereas of 80 Mantoux reactors only 6 (7.5 per cent.), showed no naked-eye evidence of tuberculosis. On the other hand, of

¹ E. Moro, *Münch. Med. Woch.*, 1908, lv., 216.

² Mantoux, *Academie des Scienc.*, Aug. 1908.

843 children with negative Pirquet reactions, 82 (9.7 per cent.), and of 386 children with negative Mantoux reactions 33 (8.5 per cent.), showed tuberculous lesions post-mortem. In view of the fact that in the presence of tuberculosis a negative reaction may be obtained (1) if there is high fever, (2) if the tubercular infection is very severe (miliary tuberculosis), and (3) if there is a modification of allergy as occurs during convalescence from measles, and since the absence of tuberculosis

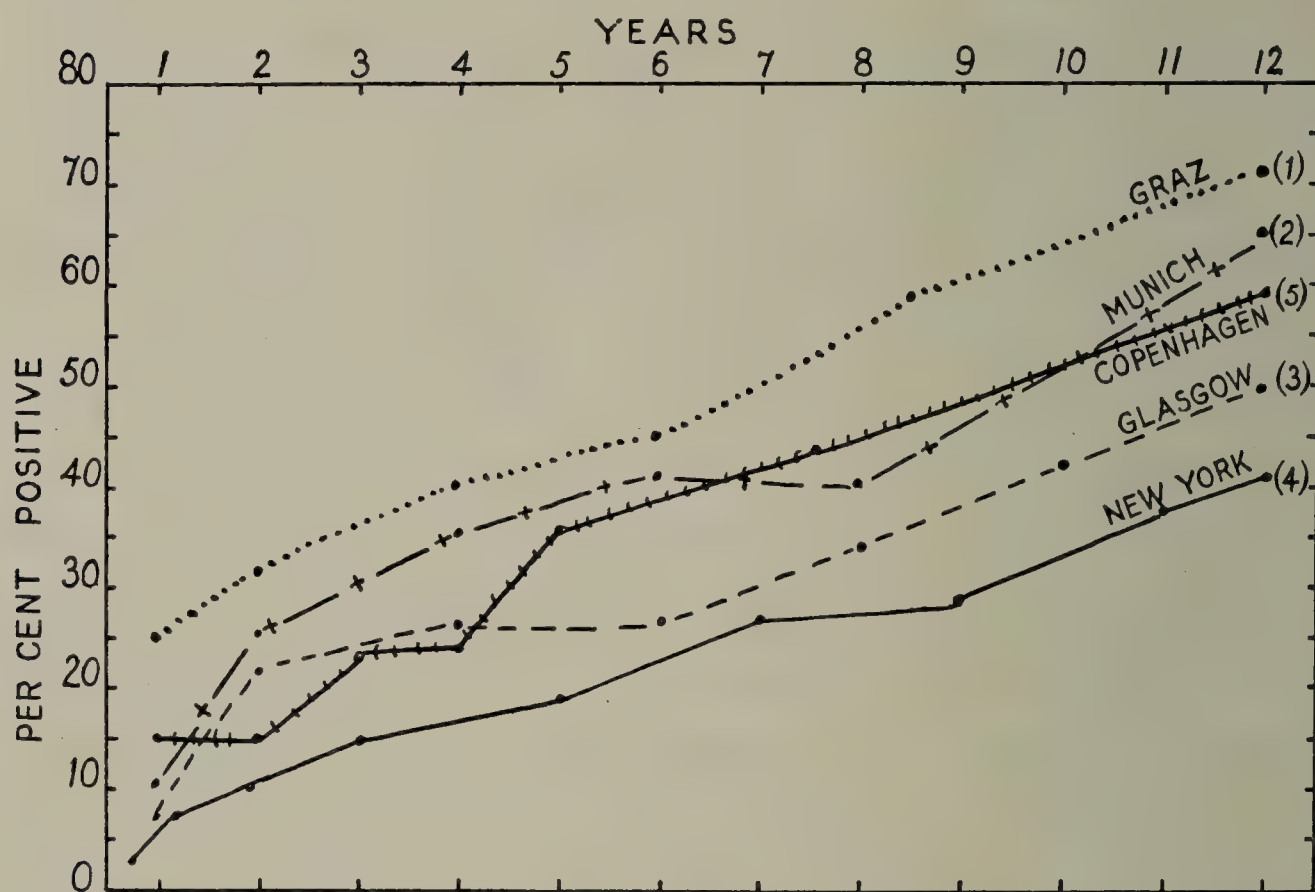


FIG 332.—Incidence of Positive Mantoux Tests in children of different ages in various centres.

- (1) Barchetti, *Arch. f. Kinderheilk.*, 1922, lxxi., 180.
- (2) Barchetti, *ibid.*
- (3) Findlay, R.H.S.C., Glasgow (941 children), unpublished.
- (4) Smith, *Amer. Journ. Dis. Child.*, 1929, xxxviii., 1137.
- (5) Krossgaard, *Acta Pædiatrica*, 1926, v., 103.

in the above observations was decided by naked-eye examination alone, it is not improbable that this test underestimates rather than exaggerates the extent of the disease.

In the above graph (Fig. 332) are shown the results of Mantoux tests in different centres. It is apparent that while there prevails a difference in the incidence of the infection in different countries there is a uniform increase from between 5 to 20 per cent. during the first year to between 50 and 70 per cent. during the twelfth year of life.

It must be appreciated, of course, that these results, just as was the case with the autopsy findings, refer to the hospital class, and, be it noted, to the sick of the hospital class of the community. No statistics regarding the incidence of the disease among the child population as a whole are available. Most authorities are agreed that in private practice positive tuberculin reactions are much less frequently obtained. Schlossmann¹ examined 105 private patients by the Pirquet cutaneous method and only obtained a positive reaction in four girls, aged 1½, 2, 5, and 6 years.

Significance of the Cutaneous Tuberculin Tests.—Although the cutaneous test is specific for tuberculosis, and is probably never obtained unless there is some tubercular infection, it must be appreciated that it does not differentiate between the human and bovine types of mischief, or between active and latent disease. All that a positive reaction as a rule indicates is that there is a tuberculous focus somewhere in the body. A negative reaction, indeed, has often more significance, since, unless in the presence of fever, during convalescence from measles, or in the presence of a very severe infection, it is almost certain proof that there is no tuberculous disease, either active or latent. Nevertheless, certain conclusions may be drawn regarding the activity or otherwise of the lesion if the age of the patient is taken into consideration. It will be admitted that the younger the patient the less time there has been for healing to occur, and hence the more likely is the mischief to be active, more especially when we appreciate that the younger the individual the less resistant is he to the tuberculous disease. Petersen and Osterfeld² record that of children reacting positively during the first year of life, 37·7 per cent. died of tuberculosis before the age of three years, while of children reacting for the first time during the second or third years, 17 per cent. and 10 per cent. respectively died before they reached the age of five years. This diminishing gravity in the significance of a positive tuberculin reaction in the later years of childhood is well brought out in a survey of the fate of a series of children sixteen years after the Pirquet tests had been performed. In 1914, Sorensen tested 5456 school children between seven and fifteen years of age; 2129 reacted positively

¹ Schlossmann, *Münch. Med. Woch.*, 1909, lvi., 398.

² Petersen and Osterfeld, *Ugeskr. Laeg.*, 1930, ii., 865.

and 3327 negatively. In 1930, it was found that of the 2129 reactors 104 (4.9 per cent.) had developed active tuberculosis, and of these 56 per cent. had died; and of the 3327 non-reactors 155 (4.6 per cent.) had also developed tuberculosis, and of these 54 per cent. had died.¹ These observations justify the generally accepted ruling that a positive reaction during the first year should be taken as meaning active tuberculosis, during the second year as possibly implying active disease, but that after the third year it is of no special significance in the absence of general or local signs of disease. As a natural corollary, it follows that all infants under one year, and even under two years, who react positively should be treated as if they had active tuberculosis. The fact that 63 per cent. of the former and 83 per cent. of the latter group survive without developing any signs of active disease is proof that much of the tuberculosis at this early age is curable. And further, these considerations indicate that it would be a wise precaution to test as a routine all infants under two years periodically, *e.g.*, every six months, and in the presence of a positive reaction place the child under the most favourable surroundings for bringing about a cure. There is no doubt that in this way much infant life would be saved.

Age Incidence of Tuberculosis.—When considering the age incidence of tuberculosis one must differentiate sharply between active and latent tuberculosis. It has been shown both in the post-mortem room (Table below) and from the results of tuberculin tests (Fig. 332, p. 948) that the incidence of the tubercular infection increases with age. In the older ages, however, much and a

Incidence of Total Tuberculosis and Healing Tuberculosis as discovered Post-mortem for Different Ages during Childhood. (Hamburger and Schluka).

Age in Years.	Per cent. with Tuberculosis.	Per cent. of Tubercular Lesions Healing.
0-1	15	0
1-2	42	0
3-4	59	10
5-6	60	40
7-10	64	40
11-14	77	47

¹ H. Ouren, *Norsk. Mag. Laegvidensk*, 1931, xcii., 139.

steadily increasing amount of the mischief is of the latent variety (Table, p. 950). The diminution of the total amount of active tuberculosis as age advances is revealed in the mortality statistics of most countries. In the chart on p. 944 (Fig. 331) are shown the death-rates at different age-periods for all forms of tuberculosis in England and Wales. This chart reveals the highest death-rate during the first and second years of life, and a steady decline till the age of puberty, with a rise thereafter reaching its height in adolescence or adult life, but not such a high level as had prevailed during the first two years. The admissions of active tuberculosis to a Children's Hospital show the same decline from the first year of life till the age of twelve years (Fig. 333).

Bovine and Human Tuberculosis.—In our first few sentences we remarked that tuberculosis was imbued with a special importance from the fact that were we to apply the knowledge we already possess much of the disease could be prevented. This refers, of course, to that variety caused by the bovine type of the tubercle bacillus. The bovine tubercle bacillus is responsible for much of the latent and non-fatal varieties of the disease. Blacklock found that in children dying of tuberculosis the human type of the bacillus was recovered in 72·7 per cent. of the cases investigated, whereas of the children in whom tuberculosis was an accidental finding at the post-mortem examination the lesion was due to the bovine type of the bacillus in 86·7 per cent.

Infection with the human type of the bacillus occurs mainly by the respiratory tract, and with the bovine type *via* the alimentary tract. Thus for the most part respiratory tuberculosis is of human origin. It is generally estimated that at

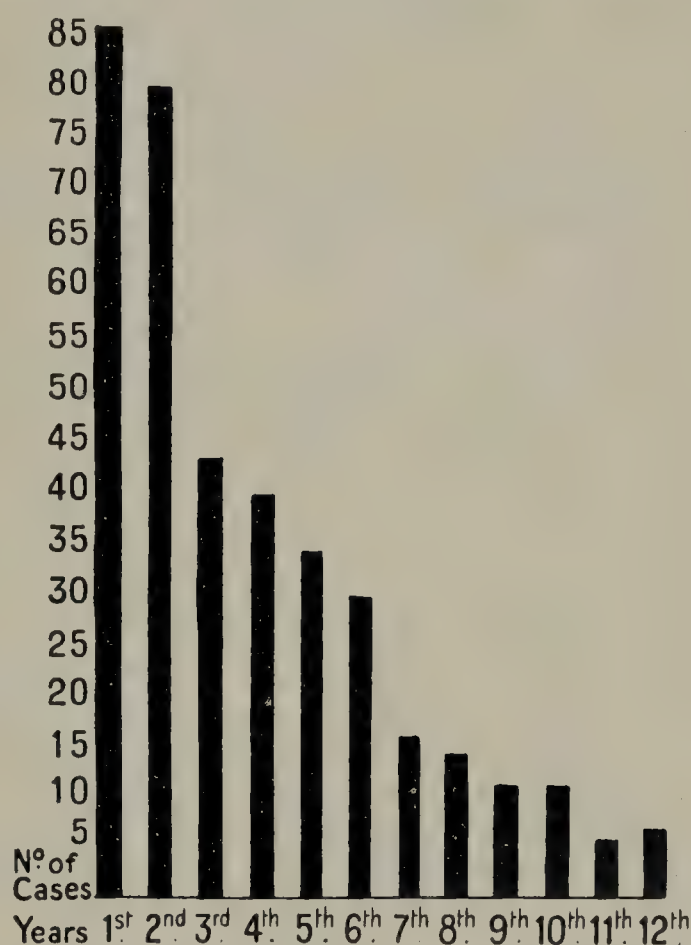


FIG. 333.—Age Incidence of Medical Tuberculosis—all Forms. (R.H.S.C., Glasgow).

least 99 per cent. of the examples of this variety of the disease are due to the human type of the bacillus. Blacklock, working with children, found that in cases with a primary lung lesion the human type of the bacillus was invariably the causative organism. On the other hand, non-respiratory tuberculosis is not infrequently (roughly 25 per cent. of the cases) caused by the bovine type of the bacillus. If latent tuberculosis were also possible of examination, no doubt even a larger proportion would be found of bovine origin. But the exact proportion of the non-respiratory examples of bovine origin varies, not only in different parts of the world but also according to the region of the body involved. These facts are revealed in the following Table compiled by Blacklock¹ and

Proportion of Human and Bovine Strains of Tubercle Bacillus isolated from Tuberculous Disease of Cervical Glands and Bones and Joints in Children.

Country.	Author.	Age in Years.	Strains Isolated from Cervical Glands.			Strains Isolated from Bones and Joints.		
			Human.	Bovine.	Bovine per cent.	Human.	Bovine.	Bovine per cent.
England . . .	Griffith . .	0 to 15	31	44	58·7	337	89	20·9
Germany . . .	Möllers . .	0 „ 16	105	54	34·0	85	3	3·4
East of Scotland	Mitchell . .	0 „ 13	18	70	88·0	23	41	61·2
„ „	Griffith and Munro	0 „ 15	7	7	50·0	16	7	30·4
West of Scotland	Blacklock . .	0 „ 12	10	18	64·3	17	9	34·6
Scotland . . .	Griffith . .	0 „ 15	4	10	71·4	20	8	28·6
Toronto . . .	Price . . .	0 „ 14	17	13	43·3	72	3	4·0

showing the relative frequency with which the bovine and human strains of the tubercle bacillus were isolated from tubercular lesions of the cervical glands and of the bones and joints. One rather anomalous and inexplicable feature in the above Table is the disproportion of bovine infection of the glands and of the joints in Germany and Toronto.

Fortunately there has been during the present century a marked and steady decline in the death-rate of all forms of tuberculosis. This fall has been more marked in the case of the non-respiratory examples of the disease, which, as we have

¹ J. W. S. Blacklock, *Edin. Med. Journ.*, 1932, xxxix., 190.

stated, is the group which possesses the largest proportion due to the bovine type of bacillus. In England and Wales, since 1914, the death-rate of pulmonary tuberculosis has fallen by 29 per cent. and of the non-pulmonary varieties of tuberculosis by 46.3 per cent. The figures for children under fifteen years of age for the city of Glasgow (Table below), which were kindly

Case-Rates and Death-Rates of Pulmonary and Non-Pulmonary Tuberculosis in Children under 15 years (Glasgow).

Year.	Pulmonary Tuberculosis.		Non-Pulmonary Tuberculosis.	
	Case-Rate per 100,000.	Death-Rate per 100,000.	Case-Rate per 100,000.	Death-Rate per 100,000.
1915 . .	123 } 145	33 } 39	336 } 340	148 } 158
1916 . .	167 }	46 }	344 }	169 }
1917 . .	167	53	330	139
1918 . .	138	56	307	137
1919 . .	115	35	268	108
1920 . .	113	35	273	102
1921 . .	137	30	259	94
1922 . .	104	30	240	90
1923 . .	92	29	284	105
1924 . .	109	27	290	94
1925 . .	84	28	260	91
1926 . .	88	22	229	75
1927 . .	78	18	235	71
1928 . .	89	23	228	76
1929 . .	85	23	215	68
1930 . .	69 } 73	21 } 20	238 }	83 }
1931 . .	78 }	19 }	208 }	70 }
Fall per cent.	49.6	48.7	35.0	51.9

supplied by Dr MacGregor, M.O.H., reveal the same tendency, but nothing like the same disproportionate decrease. The fall in the pulmonary death-rate for children under fifteen years in Glasgow amounts to 48.7 per cent., and in the non-pulmonary to 51.9 per cent. It is interesting to note, however, that while the fall in the incidence of the pulmonary examples of tuberculosis, as evidenced by the notification returns (case-rate), is practically identical with that in the death-rate for the same type, the diminution in incidence of the non-pulmonary varieties lags considerably behind the fall in the corresponding death-rate, these being 35 per cent. and 51.9 per cent. respectively.

Indeed, in the case of England and Wales, there has been for all ages since 1920 a slight increase in the notification rate of non-pulmonary tuberculosis. This does not necessarily mean that there has been an actual increase in the incidence of non-pulmonary tuberculosis, but is due in part to more complete returns, and in part to the notification of much indefinite abdominal disease erroneously as tuberculosis. That the latter factor is chiefly responsible is suggested by the analysis by Dr MacGregor of the death-rates in Glasgow for different varieties of tuberculosis according to age, in which it is shown that there has been the greatest fall of all in the deaths from abdominal tuberculosis (Table, p. 955).

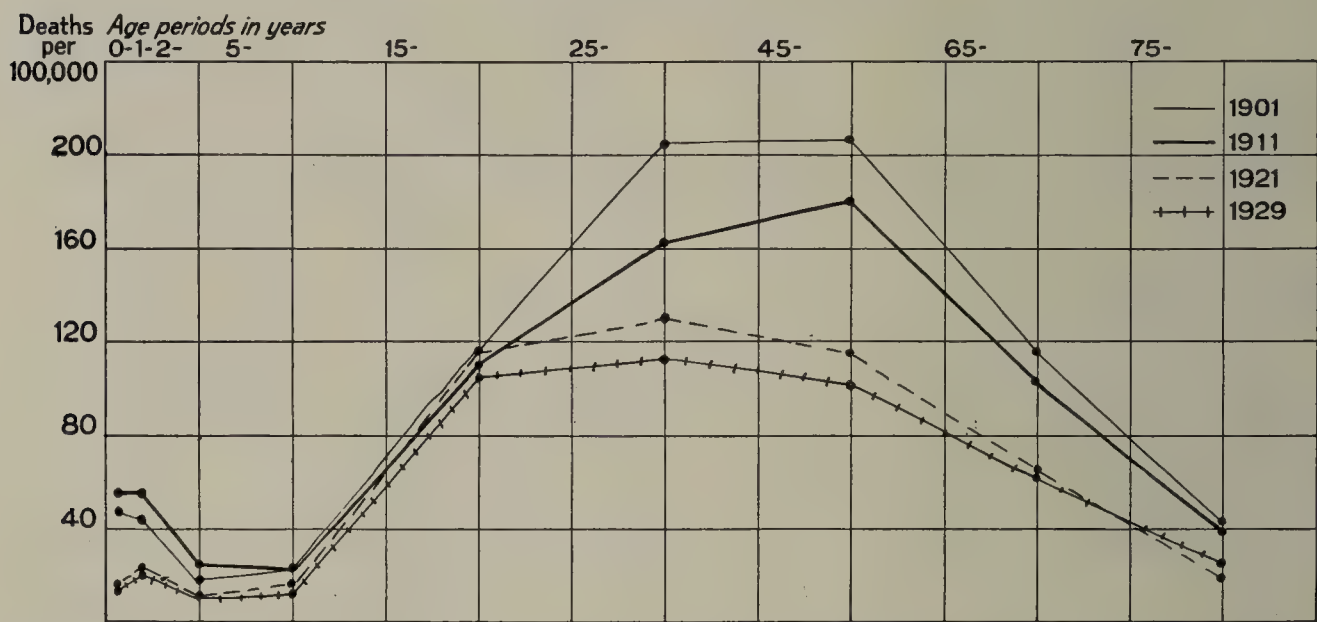


FIG. 334.—Death-Rates per 100,000 population from Pulmonary Tuberculosis at different Age-Periods for various years from 1901 to 1929 (England and Wales).

This fall in the death-rate of non-pulmonary tuberculosis is, as is to be expected, most marked during the earliest years of life. In the above chart and in that on p. 956 are plotted the death-rates in England and Wales from pulmonary and non-pulmonary tuberculosis for different age-periods during the years 1901, 1911, 1921, and 1929. For the first year of life between 1901 and 1921 the death-rate from non-pulmonary tuberculosis diminished by 78 per cent., whereas the greatest fall in the death-rate from pulmonary tuberculosis at any age (25 to 65 years) during the same period only amounted to 40 per cent. It is tempting to ascribe the greater fall in the non-pulmonary death-rate, and especially during the first year of life, to the increasing purity of the milk supply and/or to the increasing practice of pasteurisation; but the fact that the proportionate falls in the death-rates during

Non-Pulmonary Tuberculosis, Glasgow. Death-Rates per 100,000 at each Age Group.¹

Average of Years.	Tubercular Meningitis.					Abdominal Tuberculosis.					Other Forms of Tuberculosis.					Total.			
	- 1.	- 5.	- 10.	- 15.	+ 15.	All Ages.	- 1.	- 5.	- 10.	- 15.	+ 15.	All Ages.	- 1.	- 5.	- 10.		- 15.	+ 15.	All Ages.
1900-2 .	308	151	54	54	3	32	250	159	37	31	6	32	105	59	36	19	12	22	86
1910-12 .	372	165	43	43	4	32	175	89	38	19	7	23	160	61	33	22	15	25	79
1920 22 .	143	81	25	25	2	15	56	40	18	15	5	11	35	33	23	16	11	15	41
1928-30 .	100	82	29	29	3	16	20	20	8	7	4	6	29	22	7	8	9	10	32
Percentage fall	67.5	45.6	46.2	46.2	0	50	92	87.4	78.3	77.3	33.3	81.2	72.3	62.7	80.6	57.8	25.0	54.5	62.7

¹ Dr MacGregor, M.O.H., Glasgow, kindly supplied this Table.

the periods 1901 to 1911 (when pasteurisation was little practised) and 1921 to 1929 (when pasteurisation was widely

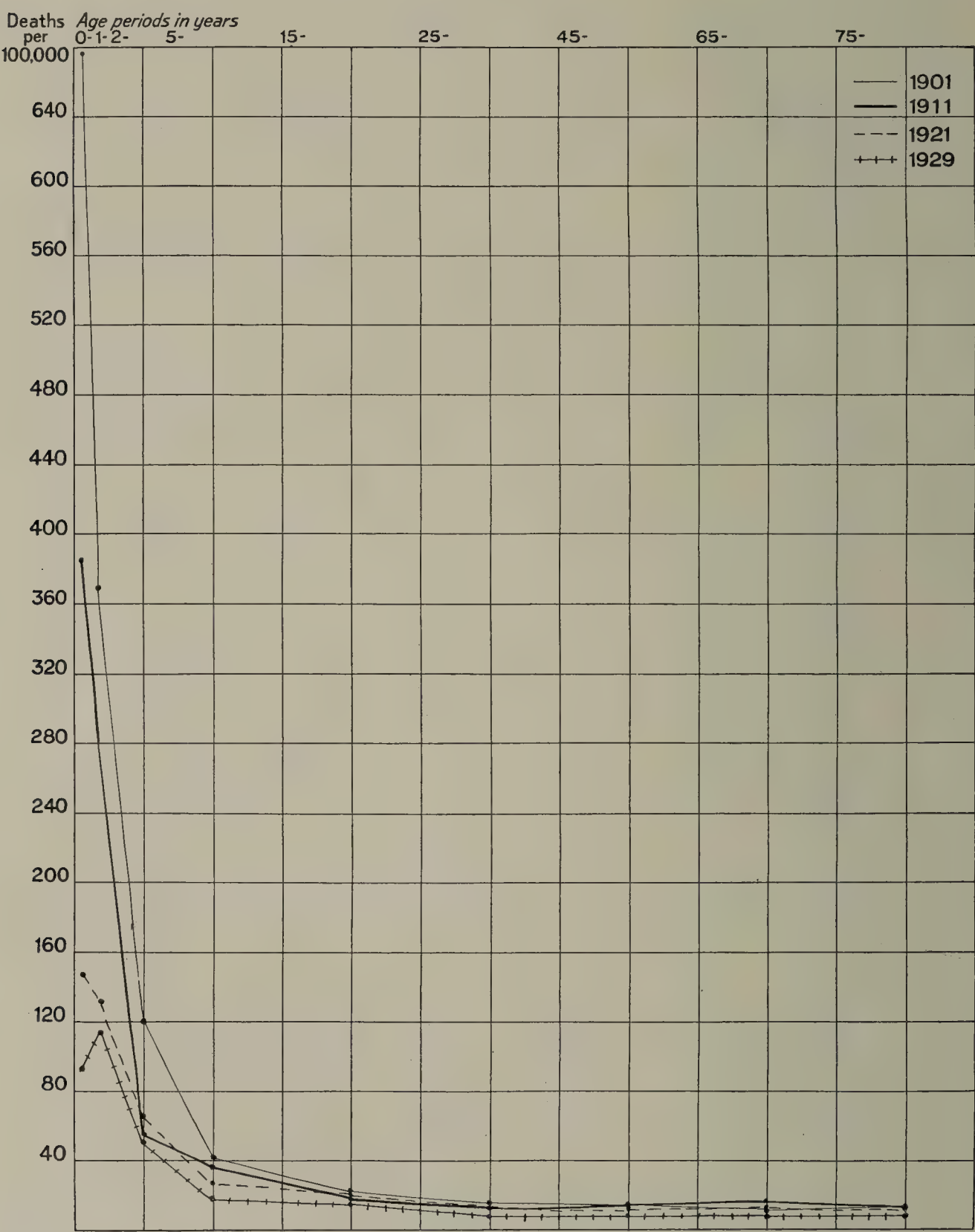


FIG. 335.—Death-Rates per 100,000 population from Non-Pulmonary Tuberculosis at different Age-Periods for various years from 1901 to 1929 (England and Wales).

practised) were 43.1 and 37.4 per cent. respectively makes one hesitate to dogmatise regarding the cause.

The absolute amount of bovine infection cannot be stated, but it is undoubtedly considerable. Recently a committee set

up by the People's League of Health¹ estimated (*a*) that about 6 per cent. of all deaths from tuberculosis are caused by the bovine type of the bacillus; (*b*) that about 2000 deaths, mostly in children, occur annually from this cause; (*c*) that at least 4000 fresh cases of bovine infection develop each year; and (*d*) that an immense amount of suffering, invalidity, and often permanent deformity is caused by this bacillus so far as England and Wales are concerned.

The source of this type of infection is of course cow's milk, and until we can obtain a pure milk supply, or only use for human consumption milk which has been pasteurised, there can be no real improvement. As a result of the already referred to inquiry conducted by the People's League of Health, it would appear that during the last ten years there has been little improvement in the purity of our milk supply. They conclude (*a*) that at least 40 per cent. of the cows in this country are infected with the tubercle bacillus, though only a minority of these are in an infective condition; (*b*) that about 0.2 per cent. of all cows are suffering from tuberculosis of the udder and are therefore probably excreting living tubercle bacilli in the milk; (*c*) that about 40 per cent. of cows slaughtered in the public abattoirs show naked-eye lesions of tuberculosis; and (*d*) that a proportion of the raw market-milk, varying in different parts of the country from 2 to 13 per cent., with an average figure of 6.7 per cent., contains living tubercle bacilli. It has been shown by work in America, and also as a result of the activities of the National Institute for Research in Dairying (Reading), and of the Hannah Research Institute (Ayr), that by the formation of herds of tuberculin non-reacting cows a milk free of tubercle bacilli can be guaranteed, but to use only such herds for our supplies to-day would cause a milk famine. With sterilised milk there is, however, no danger, and it can truly be said that were efficient pasteurisation of milk universal, tuberculosis due to the bovine bacillus could be stamped out. When we remember that the tubercle bacillus is not the only source of danger, but that streptococci, diphtheria bacilli, and the *Bacillus typhosus* may all be present and thus the cause of disease, it would appear that raw milk, whether from tuberculin-tested cattle or not, should never be used for human consumption.

¹ *A Survey of Tuberculosis of Bovine Origin in Great Britain*, People's League of Health, London, 1931.

The Primary Lesion.—It is held by many that there has frequently been no primary tuberculous lesion at the point of entrance of the bacilli, whether on the tonsil, lung, intestine, or elsewhere. It is probable, however, that Pirquet, Ghon, Dunn, and others are right in believing that a primary lesion has always existed in these localities, although it has usually been so small and has healed so rapidly that it has escaped observation in most cases. From this primary lesion the bacilli are carried to the neighbouring lymphatic glands, which generally become caseous. As the disease spreads in the glands the lymphatics become blocked, and the bacilli spread back into the lungs or intestine, or, getting into the circulation, are carried to the meninges or other serous membranes; or general miliary tuberculosis may occur.

Differences in the Distribution of the Disease in Infancy and in Later Childhood.—The two types of bacilli cause very similar manifestations; these vary, however, considerably according to the age at which the patient is attacked. In early infancy the disease is apt to take the form of a general septicæmia and to be rapidly fatal; and any local disease is likely to spread to neighbouring tissues more rapidly and certainly than in older children. In early childhood there is a greater tendency for the lymphatic glands to be severely affected; and it is only when this first line of defence is broken down that the disease is able to reach the lungs and other internal organs. The bones are also liable to be affected in young children under certain conditions. In older children the prognosis is always less unfavourable, other things being equal, as in them the disease frequently becomes localised and recovers under treatment.

General Tuberculosis.

In Infants.—When general tuberculosis occurs in babies the symptoms are usually indistinguishable in the early stages from those of ordinary marasmus or dyspepsia (p. 206). There is increasing debility and loss of weight, often with constipation and sickness, and occasionally convulsions occur. The temperature is generally normal or subnormal till the last few weeks, when there is often an irregular pyrexia, especially if the lungs are badly affected. In this case there may be increased rapidity of respiration and more or less cough. Usually there are signs

of slight bronchitis only. The spleen is often enlarged. Death may occur from progressive debility, lung disease, or meningitis.

In Older Children.—When the disease occurs in older children the symptoms are also very indefinite in the early stages, and we have chronic indigestion, loss of appetite and weight, languor, anæmia, irritability, and sleeplessness. Although nothing in the symptoms may be distinctive, there is sometimes a steadily increasing debility and cyanosis, in spite of all treatment, with an appearance of toxæmia, which is most suggestive of generalised tuberculosis. There is also often, though not always, a well-marked tuberculin reaction. Ophthalmoscopic examination reveals the presence of choroidal tubercles in a number of the cases, and sometimes there is optic neuritis. More frequently papulo-necrotic tuberculides are present in the skin, the seat of election being the trunk and thighs. These lesions appear as papules, the centres of which become necrotic, leaving a small reddish depression surrounded by a narrow halo of glossy skin.

Sooner or later some degree of fever sets in. The duration of the disease varies from a few weeks to many months. After the general symptoms have gone on for a varying time the nature of the case may be cleared up by the occurrence of serious symptoms of disease in the abdomen, lungs, meninges, or elsewhere. A considerable degree of enlargement of the spleen is commonly found.

Chronic broncho-pneumonia, with pneumococcal toxæmia, and typhoid fever are both apt to be mistaken for general tuberculosis.

Treatment.—By the time that general tuberculosis has advanced so far as to cause general symptoms it is probably incapable of being cured by any treatment; although abundant fresh air and nourishment, with attention to the child's general condition, may possibly defer the fatal issue.

Tuberculosis in the Tonsils and Naso-Pharynx.

Tuberculosis of the upper deep cervical glands often develops from a primary lesion in the tonsils or in the lymphatic tissue of the naso-pharynx. This lesion may be so small that its presence can only be demonstrated by the microscope or by inoculation experiments. It is generally found under the

epithelium near the opening of the lacunæ. In Edinburgh this form of tuberculosis is probably more commonly due to drinking tuberculous milk than to the inhalation of phthisical sputum.¹ Occasionally, when there is no tuberculous lesion in the tissue of the tonsil, bovine or human bacilli are found free in the crypts.

These facts emphasise the necessity of boiling the milk when the supply is untrustworthy, and they also show the importance of always having the tonsils and adenoids removed in cases of tuberculosis in the cervical glands.

Tuberculosis of the Cervical Glands.

Owing to the large amount of lymphatic tissue which they drain, the cervical are much more often affected than any of the other lymphatic glands; and their affection is commonest during the second, third, and fourth years of life, at which time this tissue is particularly active. They are also frequently affected during the first year and in later childhood. The infection, in Britain at least, is much oftener bovine than human—Mitchell found the bovine type of bacillus in 90 per cent. of his cases² (p. 952). The glands are infected through the tonsils, or through the other lymphatic structures in the naso-pharynx, although, as mentioned above, the lesions in these situations may be so slight and transient that it may not always be possible to discover them.

Many, but by no means all, of the children who suffer from tuberculous cervical glands have a family history of tuberculosis, or have had their resistance lowered by a recent attack of some weakening disease.

Pathology.—The bacilli may be carried by the lymphatics to the periphery of the glands; or, as happens more frequently, by the blood-stream into their centre. As the disease spreads outwards, periadenitis develops, and the glands become matted together. Finally, rupture may take place into the surrounding tissues, so that a cold abscess forms and works its way outwards, ultimately destroying the skin and causing a sinus which becomes the seat of a mixed infection. During this process adhesions take place to the adjacent muscles, to the sheath of

¹ A. P. Mitchell, *Journ. Path. and Bacter.*, 1917, xxi., 248.

² A. P. Mitchell, *Edin. Med. Journ.*, Sept. 1914, xiii., 200; John Fraser, *Clin. Journ.*, 10th and 17th Feb. 1915, xliv., 41 and 49. (See Table p. 952)

the internal jugular, and, much less commonly, to the vein itself. The glands most frequently affected are the anterior deep cervical, or tonsillar group, the submaxillary (much less commonly), and the posterior or sterno-mastoid glands. The last-named group may be infected secondarily from the tonsillar glands, or primarily from the fauces and naso-pharynx.

Clinical Features.—The general symptoms of tuberculous adenitis are usually few and indefinite; but, in some cases, the attack begins as an acute tonsillitis with an evening rise of temperature. At first the enlarged glands are discrete and painless; then they become matted together; and ultimately a local softening occurs, with or without tenderness, which ends in reddening and thinning of the infected skin. When a sinus forms the surrounding skin often ulcerates.

Diagnosis.—The trivial nature of the general symptoms, the absence of marked local tenderness, the hardness of those glands which have caseated, and the formation of cold abscesses containing no pathogenic organisms, are all characteristic of tuberculous gland disease. The absence of obvious local lesions in the scalp, mouth, tonsils, and naso-pharynx, sufficient to account for the glandular enlargement, is also in favour of this diagnosis. An acute onset of the glandular swelling is not against the diagnosis of tuberculosis, but it is a favourable sign if the glandular swelling subsides rapidly under the use of boric fomentations. In Hodgkin's disease the glands are large, numerous, and discrete, and show no softening.

Prognosis.—The condition is generally merely a local one; for it is rare to find tuberculosis of the cervical glands spreading to internal organs. When the disease extends rapidly from one group of lymphatic glands to another it is a bad sign, as it shows a low power of resistance.

Treatment.—Rest in the open air and sunshine, and nourishing diet, are important in these cases. Exposure to the X-rays for five to ten minutes at a time, every ten days or so, has been strongly recommended; and tuberculin injections seem in some cases to do good, though they are often unsuccessful.

In the vast majority of cases tuberculous glands in the neck are best treated by excision,¹ as in this way the mischief is most quickly eradicated and the consequent scarring of the neck

¹ H. J. Stiles, *Trans. 17th Internat. Congr. Med.*, London, 1913, Sect. x. (*Dis. of Child.*), 78.

infinitesimal. There are some cases, however, in which operation is not desirable. This is so in young infants with extensive disease who are judged to be unable to stand the operation; also in cases in which the disease is so widespread that no complete operation is possible; and in those in which there are numerous sinuses and much periadenitis. In cases of a mild type, in which the glands show no signs of caseation or abscess formation, operation is sometimes unnecessary. Before the operation, if periadenitis is present, it is always advisable to treat it by the use of boric fomentations; and also that the condition of the mouth, naso-pharynx, and throat should be seen to. Tonsillectomy, when necessary, often brings about a great improvement in the state of the glands. In cases of young children in whom the disease was so extensive that no surgeon would operate on them, complete recovery has been known to take place as the result of prolonged and thorough open-air treatment along with feeding up, with fixation of the head and neck by a Treves' splint or other similar apparatus, and attention to the mouth and throat.

Tuberculosis of the Bones and Joints.

A large proportion of tuberculous disease of the bones and joints met with in Edinburgh¹ and Glasgow² is due to the bovine type of bacillus (see Table, p. 952), and are undoubtedly caused by the use of tuberculous milk. The infection is carried in the blood from its site of entrance to the synovial membranes.

Pathology.—The disease in the bone may begin in the diaphysis, the articular end, the metaphysis,³ or the epiphysis. A history of some injury, usually a trivial one, is often given; and what probably occurs in these cases is that a small vessel, the walls of which have been weakened by endarteritis, is ruptured and extravasation takes place. The tubercle bacilli in the extravasated blood proceed at once to form tuberculous follicles; and,

¹ John Fraser, *Journ. of Amer. Med. Assoc.*, 2nd Jan. 1915, lxiv., 17; and especially *Tuberculosis of the Bones and Joints in Children*, London, 1914 (A. & C. Black), 6.

² J. W. S. Blacklock, *Edin. Med. Journ.*, 1932, xxxix., 190.

³ "Metaphysis" is a term recently applied to that portion of the bone which lies immediately upon the diaphysial side of the epiphysial cartilage

before long, caseation has begun in the centre of the mass and fibrosis at its periphery. If the caseation predominates the disease tends to spread rapidly (infiltrating tubercle); while, if there is much fibrosis, the disease remains localised (encysted tubercle). An injury to a bone in which there is encysted tubercle may lead to the development of the infiltrating type.

The subject of bone tuberculosis need not, however, be further considered here, as it pertains to surgery.¹

Abdominal Tuberculosis.

In this country the abdominal cavity is a very frequent site of tuberculous lesions in young children after the first year of life. Indeed, an examination of hospital statistics seems to show that until some years ago the disease was more prevalent in Edinburgh and Glasgow than in any other cities in the world.² This is, however, not the case to-day, perhaps because the milk is so generally pasteurised. In Glasgow, since 1917, at least 70 per cent. to 80 per cent. of the milk supply has been efficiently treated.

The tuberculous lesions, however, are not usually confined to the abdomen. In most instances other foci of disease are present in the thorax or elsewhere. It is also characteristic of the disease in very young children that it tends to spread rapidly to neighbouring parts, so that it is comparatively rare to find in them a pure case of peritoneal, intestinal, or mesenteric gland affection. Usually the disease in these different situations is only part of a more or less generalised infection. It is well, therefore, to regard tuberculous peritonitis, tuberculous mesenteric gland disease (*tabes mesenterica*), and tuberculous ulceration of the bowel as merely different manifestations or phases of abdominal tuberculosis rather than as separate diseases.

¹ For a full account of the pathology, diagnosis, and treatment of all varieties of tuberculous bone and joint disease, see John Fraser, *Tuberculosis of the Bones and Joints in Children*, London (A. & C. Black), 1914.

² J. Thomson and A. Dingwall-Fordyce, "On the Relative Prevalence of Abdominal and Meningeal Tuberculosis in Children in Different Countries," *Tuberculosis in Infancy and Childhood*, edited by T. N. Kelynack, London, 1908, 115.

Abdominal tuberculosis, however, takes various clinical forms, the chief of which may be enumerated as follows:—

1. *Ascites*, in which the eruption of tubercles on the peritoneum is accompanied by the effusion of fluid either into the general peritoneal cavity or into a part of it shut off by adhesions. Tuberculous peritonitis is far the commonest cause of ascites in children.

2. *Adhesive peritonitis*, in which there is more or less general glueing together of the intestinal coils, with or without suppuration or the formation of caseous plates. Not infrequently the omentum is extensively infiltrated and may become rolled up into a sausage-shaped tumour lying across the abdomen or in the left flank.

3. *Cases with symptoms of acute peritonitis*.—In some of these there is merely an unusually acute tuberculous inflammation of the peritoneum. In others the acute symptoms are caused by the rupture of tuberculous glands or ulcers with secondary infection by other organisms.

4. *Caseous mesenteric glands (tabes mesenterica)*.—Often these give rise to no local symptoms.

5. *Tuberculous ulceration of the bowel* may occasion obstinate diarrhœa, but its symptoms are often very indefinite. Generally the mesenteric glands are severely affected.

6. *Stenosis and obstruction of the bowels*, as the result of tuberculous ulceration or adhesions, are occasionally met with, and are very important to recognise with a view to their relief by surgical operation.

7. *Latent cases*.—It is not uncommon to meet with cases in which abdominal tuberculosis has run its course in so insidious a way that no illness has been suspected, until an abdominal operation or a post-mortem examination reveals the presence of peritoneal adhesions, cicatrised ulcers, or caseous glands.

Symptoms.—These vary according to the parts first and most severely affected. In the great majority of cases, however, the onset is insidious, and the first symptoms slight and equivocal. The child is noticed to be getting thinner and paler and weaker, while his abdomen is steadily enlarging. The tongue is often quite clean. The appetite is small or capricious. The motions may be very unhealthy in character, and often there is alternate diarrhœa and constipation. Sometimes there are recurrent attacks of colic, but often no pain is complained

of. The temperature may be normal, but it is often raised in the evening; and there may also be profuse evening perspirations.

Physical Signs.—The condition of the abdomen varies considerably in different cases and at different stages in the same case. When free fluid is present to any extent, the usual signs of ascites are easily made out, and in many cases nothing else can be discovered. In cases with much adhesion of the intestine, especially in young infants, extreme tympanites is not uncommonly found. Sometimes all that can be felt is a peculiar sense of resistance, along with gurgling in many cases. This is due to the coils of intestine not moving on one another as in a normal abdomen. Sometimes flat caseous masses, with either sharp or rounded edges, are easily felt just below the parietal peritoneum. When the omentum is involved there may be dullness to percussion in the umbilical region or it may be felt as an elongated and rounded mass adherent to the abdominal wall and lying across the abdomen above the umbilicus or in the left flank. There is often, sooner or later, a rise of temperature. When suppuration begins the abdomen may become tender; and in time the umbilicus and its neighbourhood become reddened and prominent. Later, the pus makes its way to the surface in this situation.

Caseous glands, if numerous, often form large hard masses which are easily palpated. If the abdominal muscles are rigid, however, even large masses may escape detection unless an anæsthetic is administered. A rectal and bi-manual examination is generally advisable. Occasionally an abscess connected with a gland, or with an intestinal ulcer, bursts into the peritoneal cavity. When this occurs symptoms of acute peritonitis are set up and the case may be indistinguishable from one of appendicitis or septic peritonitis. In extensive ulceration of the bowel there may be considerable rigidity and tenderness of the abdominal muscles.

Stenosis of the bowel leads to distension of the portion of intestine above the obstruction, with hypertrophy of its walls. When this occurs there are often recurrent attacks of severe abdominal pain. During these the coils of hypertrophied and dilated bowel stand out prominently on the surface of the abdomen, and its peristalsis is very noticeable (Fig. 336). Loud gurgling may also be heard. These symptoms are characteristic and should not be overlooked, as they indicate a serious con-

dition which may require a surgical operation for its relief. In subacute and chronic cases of abdominal tuberculosis there is, not infrequently, tuberculous affection of the testicle and spermatic cord.

A type of case often occurs in well-nourished, previously healthy children, which is apt to pass unrecognised. In this, feverish attacks of a remittent type lasting two or three weeks recur at intervals of a few months. No other distinctive symptoms are present beyond abdominal distension, and the



FIG. 336.—Abdominal Tuberculosis with Intestinal Stenosis. (Boy of 2 years.)

first attack, at least, is apt to be diagnosed as a mild case of enteric fever. The real nature of the illness becomes evident later when it has occurred more than once. In each of these attacks there is probably a fresh, but limited, eruption of tubercles on the peritoneum, and, as the symptoms subside, an area of new adhesions is left which, in time, leads to the characteristic sensation on palpation. These cases, when early and energetically treated, generally recover satisfactorily and permanently.

Diagnosis.—In most cases this is easy, except in the early stages. Suspicions of the tuberculous nature of the case are strongly confirmed if there are other tuberculous lesions present; likewise, if there is a family history of tuberculosis, if a phthisical person has been associating much with the child, and especially if the patient has been having unboiled milk. The obstinate continuance of diarrhoea in a young child in spite of careful treatment, along with a hectic temperature, may arouse suspicions of tuberculous disease, but does not prove it. There are no special features characteristic of tuberculous diarrhoea that can be easily recognised. A negative Mantoux reaction would help in eliminating tuberculosis. A positive reaction would reveal the presence of tuberculous infection, but it would not necessarily point to this being the cause of the symptoms (see p. 949).

Prognosis.—When the abdominal condition is not complicated by the presence of severe tuberculosis elsewhere, when it is not in an advanced stage, and when there is no previous cachexia, the chance of recovery under proper treatment is very considerable. Thoroughly energetic treatment, begun fairly early and consistently persevered in, is often surprisingly successful. We have seen many cases in which large hard masses of tuberculous glands entirely disappeared after months, or perhaps years, of careful treatment, so that even under chloroform they could no longer be detected. Recovery, indeed, need scarcely ever be despaired of, except in advanced and cachectic cases in young infants. As a rule, it may be said that the older the child the better is the prognosis.

Treatment.—In this, as in most other forms of tuberculosis in children, if we are to get the best results there must be what Osler calls “masterful management” of the case in the early stages. Thorough-going and intelligent open-air treatment must be insisted on, and no half-measures allowed. So long as there are any acute symptoms the patient is better in bed, but his bed should be where it can be lifted easily out into the sunshine. Exposure of the abdomen to the sun’s rays is beneficial. The important point to remember in connection with open-air treatment is that it should be prolonged—for a year or even two years or longer—until there is every probability that the tuberculous foci have become fibrosed and encapsulated. Any place which is open and favoured by much sunshine is suitable—this is more likely at the sea-coast than in the country. During the summer any region of the British Isles is satisfactory, but during the winter the coast round the Moray Firth in Scotland, or along the south coast of England, is mild enough for the carrying out of open-air measures.

In addition to open-air treatment, every possible means must be used to increase the child’s nutrition without disturbing his digestion or irritating his bowels. All indigestible articles must, of course, be forbidden. Starchy foods have to be given in strict moderation. The more milk and cream the child can digest the better, but, if there is flatulent distension, or if curds appear in the motions, the milk should be peptonised and the amount carefully restricted. Minced underdone meat, raw-meat pulp, raw-meat juice, eggs in various forms, fish, plasmon, and somatose are of great value. When the appetite is not

good alcohol is often beneficial, and either brandy and egg mixture or port wine may be given with advantage.

Cod-liver oil, iodide of iron, creosote, and arsenic are all useful at times, provided they can be given without upsetting the digestion or lessening the appetite. The application to the surface of the abdomen of iodoform ointment (10 per cent.) or of mercurial ointment is said to do good in some cases. Should the fluid in an ascitic case increase so as to embarrass the breathing, or should it remain for more than a fortnight without diminishing, it is generally advisable to draw it off with a Southey's tube or small trocar, unless a surgical operation has been determined on. Injections of tuberculin are held by some to be useful.

On the place of surgery in the treatment of this disease there is still considerable difference of opinion. All are agreed that an operation may be necessary when abscesses form, and is always called for when stenosis is present to an extreme degree. It is also generally held that when the peritoneal cavity is to a large extent obliterated by adhesions, surgical operation is dangerous and not likely to benefit in most cases. Surgery would seem to have its special value in the early and late cases. The removal of tuberculous mesenteric glands is very rarely possible or advisable, unless the masses are large and isolated, as, *e.g.*, in the region of the cæcum or splenic flexure. When suppuration occurs, the abscess, which always tends to point at the umbilicus, should be allowed to evacuate itself spontaneously; or at least it should not be incised until the reddened skin over it is on the point of giving way. Any attempt to open the abscess while it is still deeply situated is inadvisable, because of the great risk there is of rupturing the diseased and adherent bowel.

The chief difference of opinion refers to those cases in which the peritoneum, though studded with tubercle, is not yet firmly adherent, and especially to cases of tuberculous ascites. Some years ago a simple free incision into the peritoneal cavity, with evacuation of any fluid it might contain, was regarded as greatly increasing the child's chance of life. Many cases make a satisfactory recovery after this simple operation. There has, however, always been difficulty in determining to what extent the improvement was attributable to the surgical proceeding, because the type of case which does best after it is exactly that

which most often recovers under purely medical treatment. Several competent observers have expressed strong doubts as to whether the opening of the peritoneum does any good whatever, and have suggested that it is in spite, rather than because, of the operation that the children recover.

Although formerly too much was expected from laparotomy in tuberculous ascites, it is, nevertheless, decidedly beneficial in suitable cases. When thorough medical treatment, energetically carried out, has failed to arrest the symptoms, and the disease is showing no signs of abating, rapid and continued improvement often follows incision, especially in older children and adolescents; and the risk of the operation is exceedingly small. The number of young infants benefited by laparotomy is much smaller and the risk in them is much greater. Implication of the pleura and even a slight lung affection are not contraindications to operation. Whatever the explanation may be, it is, we believe, the general experience, as well as our own, that the results of removing the tuberculous ascitic fluid with a trocar and cannula are not nearly so good as those of simple incision.

When there are clear indications of intestinal obstruction from stenosis or compression of the bowel a surgical operation is urgently called for, as has been already mentioned.

Thoracic Tuberculosis.

The modern conception of pulmonary tuberculosis has imbued the disease during childhood with the greatest significance. Nowadays it is generally taught that most intra-thoracic tuberculosis dates from early life. The tubercle bacillus entering *via* the air-passages is stated to give rise in the first instance to a minute lung focus (Ghon's tubercle), whence the disease spreads to the adjacent lymph glands. This primary focus, and the early bronchial gland mischief as well, are believed, however, not infrequently to undergo healing and to become calcified, and as such to be quite often easily identified both anatomically and radiologically. Nevertheless, because they harbour living tubercle bacilli, these foci remain a constant menace and may light up at any future time should favourable circumstances arise.

That a primary focus in the lung can be observed in much of the fatal intra-thoracic tuberculosis of childhood is undoubted,

but in our experience it is seldom healed. It can, we think, be laid down as a general law that pulmonary tuberculosis during childhood does not heal but is almost invariably fatal. Dr Blacklock,¹ working at the R.H.S.C., Glasgow, only found evidence of healing of a tuberculous focus in the lung on three occasions during the course of 1800 post-mortem examinations on children under twelve years of age. In no case was the healing complete. In these three children, who varied between three and nine years old, the pulmonary focus was an accidental finding, and in no way responsible for the fatal result. In all the cases (145) in which tuberculosis was the cause of death, and in which a primary lung focus was identified, there was absolutely no suggestion of healing. It is also interesting to note that Blacklock found the mediastinal glands infected in 25 children in whom no primary lung focus could be discovered. Of 16 of these latter which were submitted to tests in order to discover the strain of the etiological organism, 82 per cent. were caused by the human type of the tubercle bacillus. This is a proportion quite different to that present in the case of cervical and abdominal gland tuberculosis, in which Blacklock found the bovine type of bacillus in 64.3 and 86.7 per cent. respectively. It would thus seem reasonable to conclude that, just as occurs in abdominal tuberculosis due to ingestion, so in infection of the mediastinal glands by inhalation, the tubercle bacillus may leave no trace of its portal of entry. These findings of Blacklock are thus rather against the infallibility of the Ghon doctrine. Moreover, if such were really the course of events, it has always seemed to us rather anomalous that the primary lung focus should be so frequently in the lower lobe, as is generally admitted to be the case, and that active pulmonary tuberculosis, especially in later life, should show such a predilection for the upper lobe.

Radiography of the Chest.—As to-day an opinion not only regarding the presence of intra-thoracic tuberculosis, but also concerning its exact nature, is based to such an extent on the radiogram, a few words on this subject would seem not inappropriate. In the first place it must be appreciated that the lung fields even in the young infant are never devoid of shadows. The pulmonary tissue contains in the form of stroma much muscle, fibrous and elastic tissues, in addition to blood vessels

¹ J. W. S. Blacklock, *Proc. Roy. Soc. Med.*, 1932, xxv. (Path. Sect., p. 11).

and bronchi. Since all these structures vary in amount, as also the angle at which they are photographed, and as the density of the radiogram depends on the hardness of the tube and the length of the exposure, considerable variations within the range of normality are encountered. It must also be borne in mind that, whatever the nature or cause of the condensation of the pulmonary tissue, an interruption in the passage of the X-rays is caused with a resultant shadow, so that a particular shadow does not necessarily signify a special diseased process. In fact, it can be laid down as an absolute law that no radiographic picture of the chest is pathognomonic of any one diseased process. The radiogram can only be correctly read along with the physical examination and clinical history of the patient.

The amount of shadow that is compatible with normal thoracic contents was some years ago the subject of investigation by a committee of clinicians and radiologists appointed by the National Tuberculosis Association of U.S.A., and the following conclusions were formulated¹:—

(1) The normal chest of the child from the roentgenological standpoint is subject to such wide variations within normal limits as to be beyond the possibility of exact description.

(2) The conglomerate shadow, commonly called the hilum shadow, when found lying entirely within the inner third or zone of the lung, can be disregarded (or regarded as normal), except where it is made up of a solid mass of homogeneous shadow giving undoubted evidence that it represents a growth or mediastinal pleurisy. [Such a picture, but caused by massive collapse of the lung, is represented in Fig. 196, p. 630.]

(3) Calcified nodes at the root of the lung, without evidence of lung disease, are of no significance except as possible evidence of some healed inflammatory condition, possibly but not necessarily tuberculous. They are a common finding in normal chests.

We ourselves would go further than the American writers do in their third conclusion. Many of the small dense shadows (so-called calcified nodes) at the roots of the lungs, or throughout the lung fields, are not only of no significance, but are most certainly not due to calcified deposits at all. This can be demonstrated any day in the post-mortem room. In the experience of one of us (L. F.) calcification of lymph glands of such a degree that they can be recognised radiologically is

¹ *Trans. Eighteenth Ann. Meeting Nat. Tub. Assoc.*, 1922.

rare under the age of eight years, and even in older children is usually limited to the cervical and mesenteric groups. Calcification of a tubercular focus, at least in the early stage, gives rise to a characteristic crescent-shaped and not to a small, dense, globular-shaped shadow in the radiogram, and yet it is the latter type of shadow which is most frequently observed and generally considered to be due to the presence of lime deposit. A recent study of the same question at the Brompton Hospital would seem to confirm this view.¹ In the report of this work it is stated that as almost 50 per cent. (22 out of 51) of the children who were considered to have radiological evidence of calcified glands did not react to tuberculin it would appear that "the radiological criteria in this respect were not sufficiently strict," and the authors admit that a blood-vessel or other structure viewed "end on" may be mistaken for a calcified nodule.

Whatever be the truth regarding the rôle of the Ghon focus in the pathogenesis of intra-thoracic tuberculosis, clinically the disease may declare its presence in the mediastinum, in the lung, or in the pleura, and the particular situation depends in no small measure on the age of the individual and the geographical situation in which the disease is contracted. There is no doubt that age is a factor of importance in this direction. While disease of the lung is *par excellence* a manifestation during infancy and early childhood, disease of the pleura is pre-eminently met with during adult life (p. 633). The variation not only in the incidence of a particular disease but also in its clinical picture in different parts of the world are also points which we do not sufficiently recognise, and in no disease is this more true than in the case of tuberculosis. In Toronto, for example, mediastinal tuberculosis—we do not mean simply enlarged glands—is one of the most frequent pictures of intra-thoracic tuberculosis in childhood, whereas in Glasgow and the West of Scotland it is one of the rarest. This variation in the clinical picture in different countries is also observed with regard to the mortality of pulmonary tuberculosis. In Scotland, and in England too, pulmonary tuberculosis during childhood is almost invariably fatal, whereas in the Netherlands it is said that even widespread mischief is not infrequently recovered from.

Mediastinal Tuberculosis.—In most if not all examples of intra-thoracic tuberculosis there is disease of the mediastinal

¹ Dorothy J. Dow and W. E. Lloyd, *Brit. Med. Journ.*, 1932, i., 701.

glands. In some instances this may be, as mentioned above, the sole seat of tuberculosis in the chest, and since in a certain proportion of these cases (18.7 per cent. according to Blacklock's findings) the bovine type of the tubercle bacillus is the etiological organism, the disease has probably spread from the cervical or abdominal glands. Without doubt tuberculosis of the mediastinal glands is a very common type of the disease, and, while it is usually symptomless, and in the main gives rise to no more illness than is the case with much of the abdominal gland tuberculosis, it is, especially during childhood, a constant menace and is responsible for a considerable proportion of the fatal examples of tuberculosis. There may result (*a*) a backward spread into the lung tissue with pulmonary tuberculosis, (*b*) rupture into a vessel with a consequent miliary tuberculosis, or (*c*) rupture into a bronchus with the production of a caseating broncho-pneumonia (galloping consumption); (*d*) in some cases without any involvement of the lung tissue the glandular enlargement may become truly enormous and simulate tumour formation. It is this last variety (Fig. 337) which we have stated is so common in Canada.

If the disease is latent there may be no symptoms, but even if the disease is active, and there are present general toxic manifestations (fever, malaise, and wasting), it may not be possible to localise the seat of the mischief as being in the mediastinum. This is because the glands do not give rise to any pressure phenomenon, or are too small to be detected either by physical examination or radiology. Sometimes, in consequence of pressure on the trachea, a peculiar paroxysmal cough is produced, and on rare occasions pressure on the œsophagus has caused dysphagia. Distension of the superficial veins of the thorax or in the neck and in the arms may be apparent. If the glandular mass is large, dullness to percussion over the sternum or vertebræ, or in the parasternal and paravertebral areas, may be detected. D'Espine's sign and the Eustace Smith phenomenon have been, in our hands, of little help. Radiology in the vast majority of cases is of comparatively little value. Correlation of the radiogram with the post-mortem findings has convinced us of the extreme difficulty of recognising any particular shadow as due to an enlarged gland. Unless the glands are very much enlarged and extend laterally, it is impossible to differentiate between them and the general

mediastinal tissues. In some cases, however, they are quite apparent (Fig. 338) and, as in the Toronto example illustrated below (Fig. 337), the picture is most striking. There is no doubt that it is the geographical variation in the distribution of this

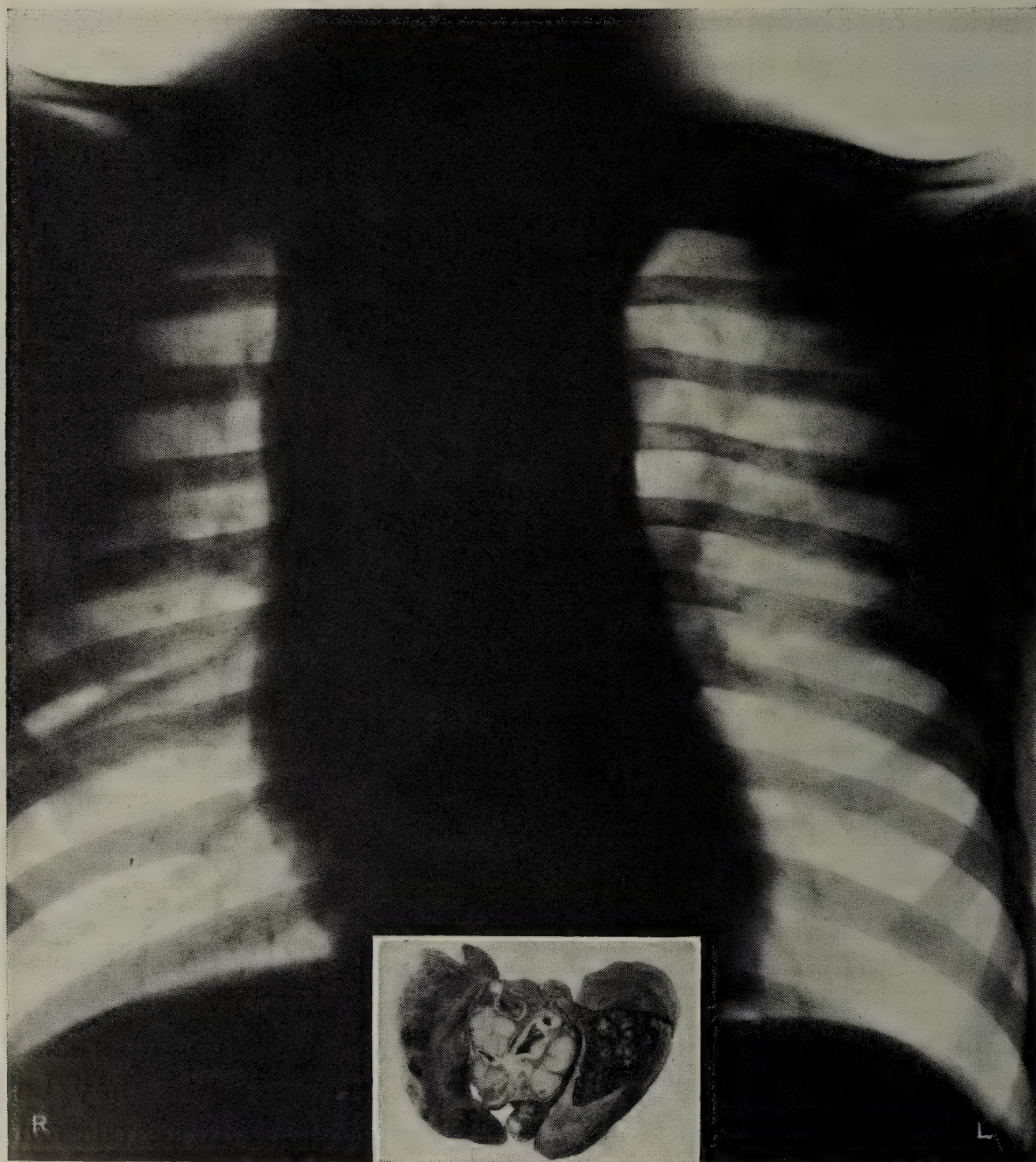


FIG. 337.—Skiagram of Chest in case of Extensive Tuberculosis of Mediastinal Glands. (Boy aged 10 years.) In small inset photograph is seen the conditions post-mortem. (Photographs kindly supplied by Prof. Alan Brown, Toronto.)

variety of tuberculosis which is in great part responsible for the diversity of views on the matter.

Pulmonary Tuberculosis.—Pulmonary tuberculosis is pre-eminently a disease of infancy and early childhood. There are more deaths from pulmonary tuberculosis during the first two

or three years of life than during any other age-period. It is, moreover, an acute disease and is, as a rule, or rapidly becomes, a generalised infection of the lungs.

Acute caseating tuberculous broncho-pneumonia is usually met with during the first year of life. Due to contact with some

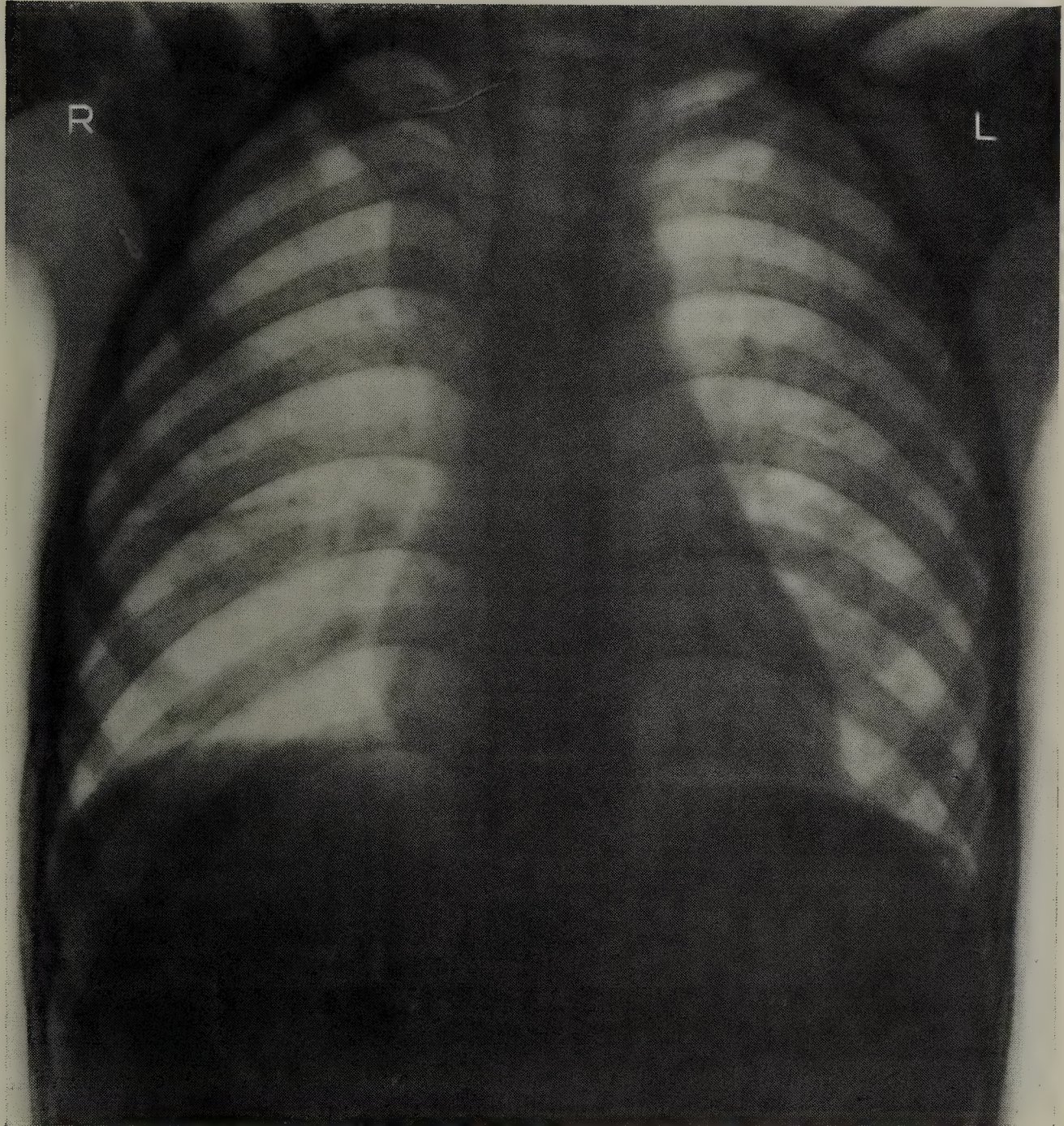


FIG. 338.—Skiagram showing Enlargement of Tracheo-Bronchial Glands on Right Side. (Girl aged $6\frac{1}{2}$ years.)

phthisical individual (the mother, the father, or the nurse) infection results and gives rise in the first instance to a localised lesion which may involve the greater part of one lobe (Fig. 339). Diffuse caseation with cavitation rapidly supervenes and by bronchial spread a generalised tuberculous broncho-pneumonia soon terminates the picture (Figs. 340 and 341). The first and

often the only symptom is a troublesome cough. There may be no fever, and nutrition is usually maintained so long as the child drinks well and the mischief is more or less localised, so that the disease is frequently far advanced before advice is sought. In the early stage physical examination reveals

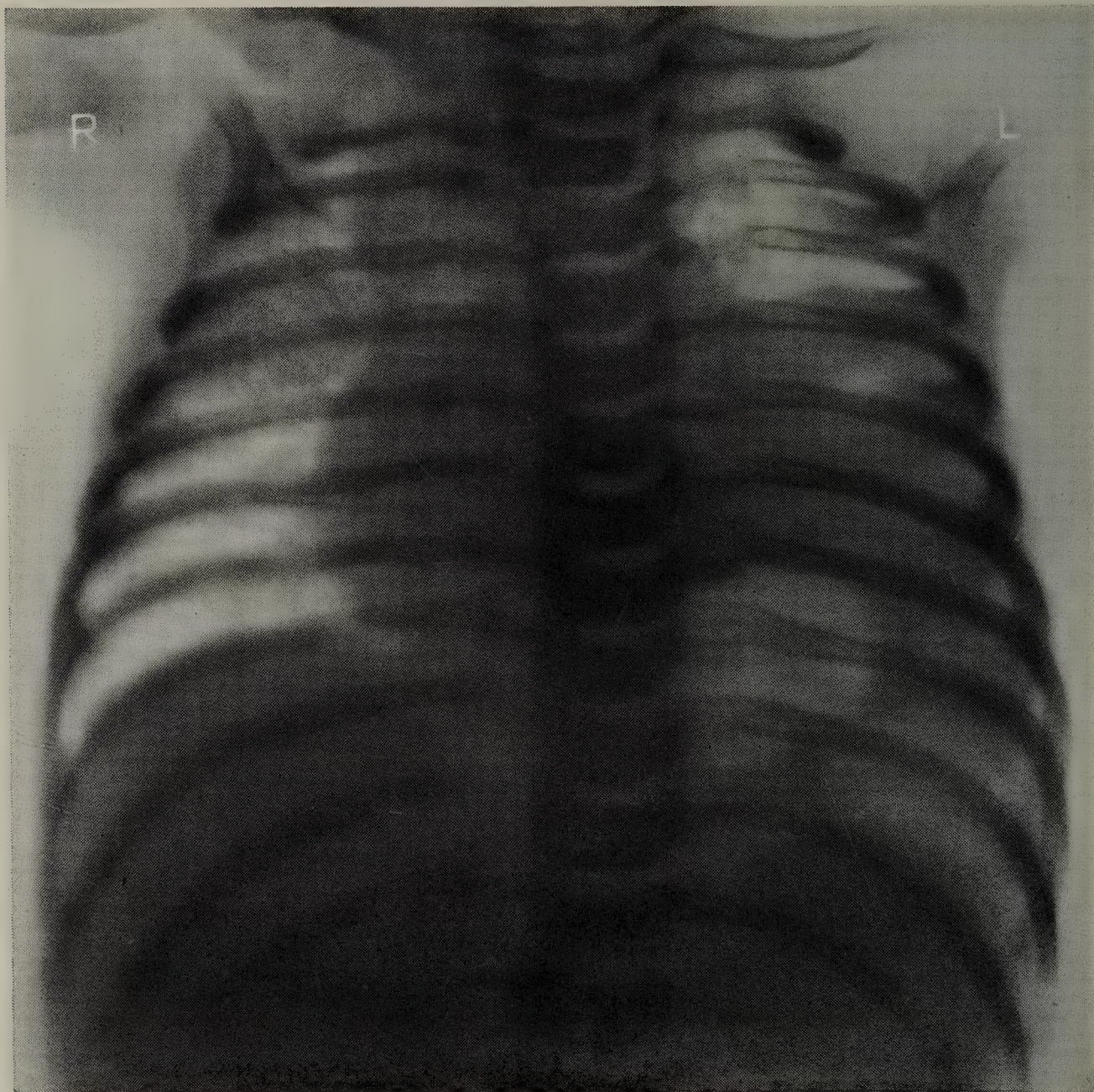


FIG. 339.—Skiagram of Tuberculous Broncho-Pneumonia of Left Lower Lobe with cavity formation in child aged three months.

dullness and tubular breathing limited to the area of the lung involved, and, if softening has taken place, moist subcrepitant râles in addition. At this stage X-ray examination may only show a localised shadow with nothing characteristic of a tuberculous lesion, and the condition may be mistaken for a simple pneumonia. There may be, however, appearances suggestive of cavitation. Examination of the sputum should never be omitted. Sputum can always be obtained by irritating the



FIG. 340.—Skiagram of Tuberculous Broncho-Pneumonia at Right Apex with generalisation throughout lung. (Girl aged 11 months: skiagram taken 18.8.27. Same case as Fig. 341).



FIG. 341.—Skiagram of Tuberculous Broncho-Pneumonia at Right Apex in girl aged 11 months. (Same case as Fig. 340. Skiagram taken 22.8.27). Note breaking-down of focus at right apex and increase in size of nodules throughout the rest of the lungs.

pharynx with a piece of gauze on the finger. This manœuvre induces coughing and the secretion is swept out of the mouth with the finger before it is swallowed, transferred to a slide and examined in the usual fashion. Examination of the stomach washings or of the fæces for tubercle bacilli has been recommended, but we have never obtained a positive finding by either of these methods when it was not more easily got by direct examination of the sputum recovered as described above. A positive Mantoux reaction is a great help in diagnosis, since at this age (the first year of life) a positive result usually spells active disease. However, if the child is highly fevered, or in the very latest stage of the disease, a positive reaction may not be obtained.

The correct diagnosis should not be long delayed, since comparatively soon there develops dissemination throughout the air-passages with the production of widespread broncho-pneumonia, or dissemination by the blood stream with a resultant miliary or general tuberculosis. Physical examination of the chest may not disclose any extension of the mischief, but X-ray examination will reveal breaking-down and a diffuse fine or coarse mottling of the lungs ("snow-storm" picture) (Figs. 340 to 342). When the mischief is widespread throughout the lungs, cyanosis is a prominent feature. Dissemination by the blood stream may determine the presence of tubercles in the choroid, or of papulo-necrotic tuberculides in the skin. This type of the disease in our experience is invariably fatal and uninfluenced by any treatment, although the French have recorded recoveries from artificial pneumothorax therapy.

After the age of one year the type of pulmonary tuberculosis most frequently encountered is a diffuse broncho-pneumonia or subacute miliary tuberculosis. In the average example of acute miliary tuberculosis terminating with tubercular meningitis the lesions in the lung are neither large enough, nor numerous enough, either to give rise to symptoms or to be detectable by either physical examination or radiology. But if the miliary mischief is less acute (subacute), and the meningeal involvement is long delayed, the pulmonary lesions may be possible of detection. In addition to the usual symptoms of toxæmia and fever there are, if the lung is seriously involved, cyanosis, cough and dyspnoea. On physical examination of the chest, however, little more abnormal than an occasional râle may be detected,

but an X-ray examination will reveal generalised stippling ("snow-storm" picture) (Fig. 342). In the very early stages it may be difficult to be sure of the mottling, but in these circumstances a subsequent X-ray examination after an interval of a week or ten days will usually leave no room for doubt. If the mottling

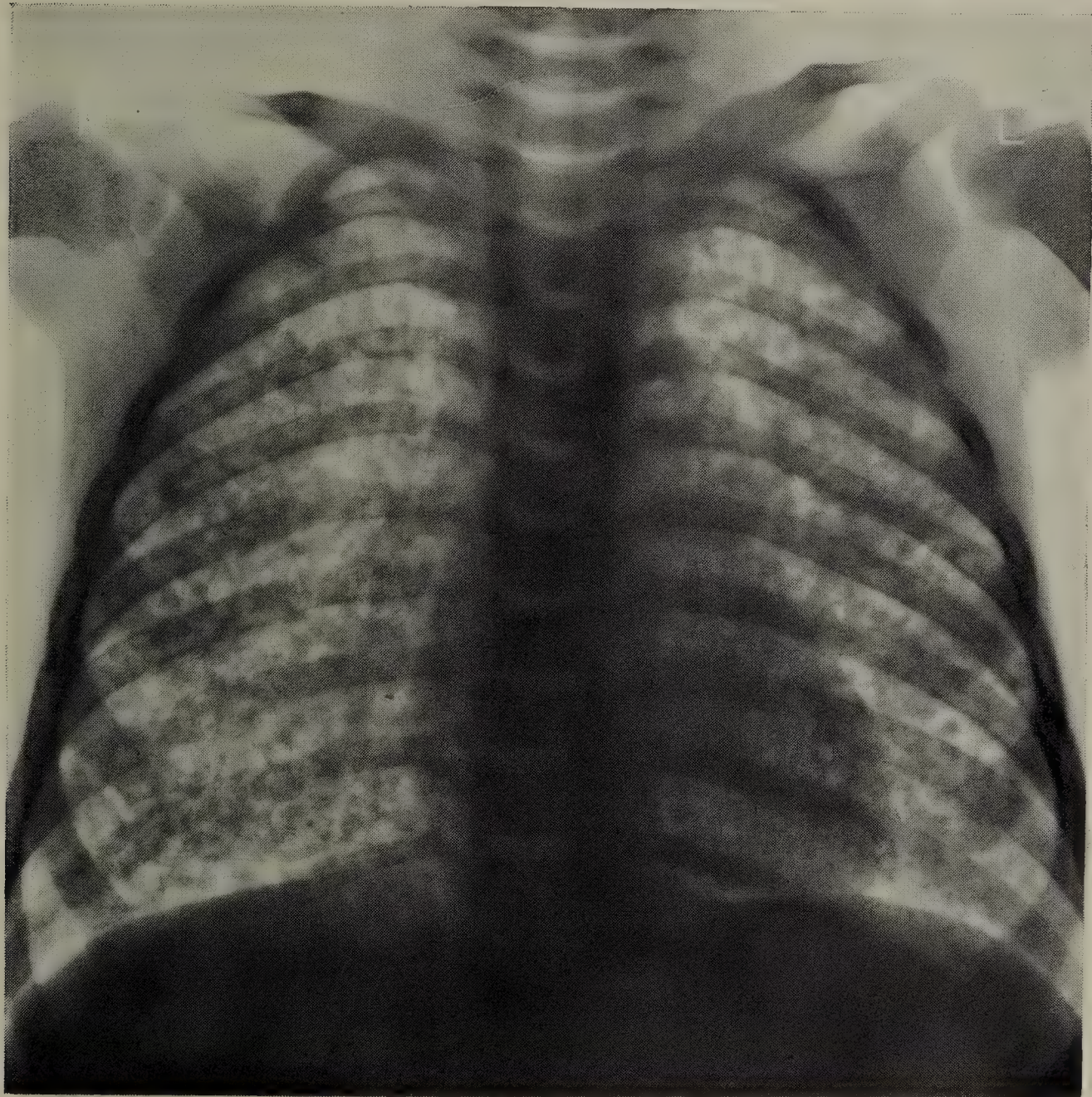


FIG. 342.—Skiagram in Subacute General Tuberculosis (miliary or broncho-pneumonic) of both Lungs, giving the typical "snow-storm" picture. (Boy aged 2 years and 1 month.)

is due to tubercles the shadows steadily increase in size and number. As a rule, however, the X-ray appearances are far in excess of what had been anticipated by the symptoms or physical signs. It may be, indeed, only by radiography that tuberculosis is first suspected. In tuberculosis of the lungs the X-ray appearances are almost invariably in excess of the physical signs, which is the reverse of that which obtains in

simple pneumonia. Congenital bronchiolectasis may give rise to a picture closely simulating miliary tuberculosis, but in this condition the shadows remain constant in size and the general picture does not vary even when observed over long periods (Fig. 343). Sometimes in the normal lung the "snow-storm"

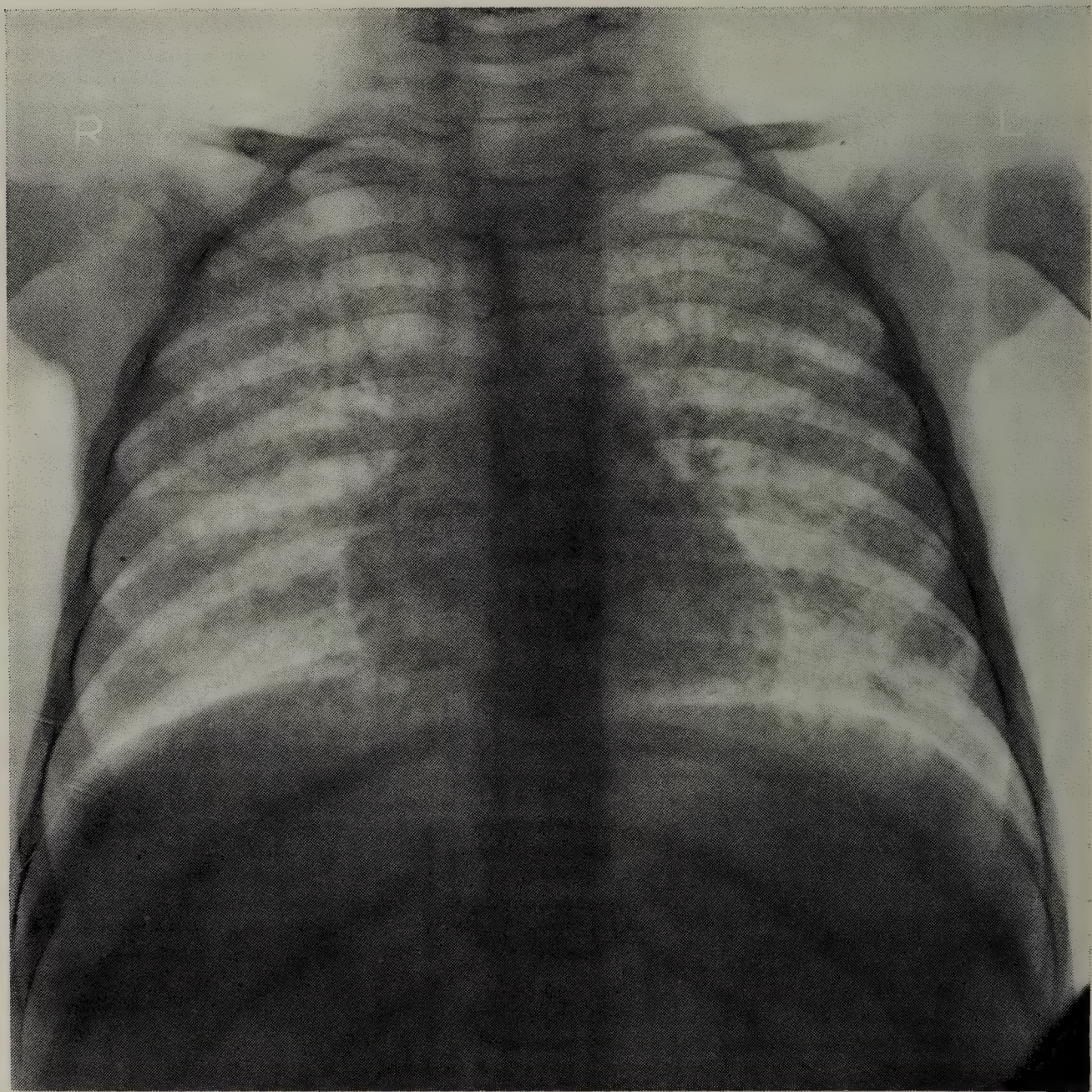


FIG. 343.—Skiagram in case of Congenital Bronchiolectasis simulating the "snow-storm" picture of general tuberculosis. (Boy aged $2\frac{1}{2}$ years—diagnosis confirmed post-mortem.)

picture is suggested. Careful scrutiny will reveal that this is due more to a network of shadows than to discrete pin-head or millet-seed-sized opacities: moreover, just as in the case with bronchiolectasis, there is a constancy in the condition from time to time, as it is not due to a proliferative lesion. It would seem that this appearance in the normal lung is consequent on an increased density of the stroma.

Sputum examination in this variety of the infection is often of little help, because, until the later stages, the lesions may be closed and bacilli do not enter the air-passages. In the later stages, however, and especially in the subacute broncho-pneumonic form, there is usually some degree of breaking-down and tubercle bacilli can be discovered in the pulmonary secretions.

Chronic pulmonary tuberculosis, as met with in the adult, is not a common disease during infancy and early childhood. As we have already remarked, pulmonary tuberculosis in childhood is an acute disease. In practically every child in whom we have obtained tubercle bacilli in the sputum, and this holds good both for the West of Scotland and London, death has supervened within the period of four years and usually within one year of the onset of the illness. Chronic pulmonary disease in childhood, whether there be evidence of tuberculosis in some other part of the body or not, is usually unresolved pneumonia or bronchiectasis (p. 641). Indeed, the duration of the illness is a most valuable diagnostic aid in differentiating between tuberculous and non-tuberculous pulmonary disease. It is only after ten years of age that chronic pulmonary tuberculosis occurs with any frequency, just as it is only after this age that cure or, at least, arrest of the condition can be expected.

Tuberculosis of the pleura, as evidenced by pleurisy with effusion, may be met with at any age from the first year of life. Unlike tuberculosis of most other regions of the body, it becomes increasingly instead of decreasingly frequent as age advances.¹ Indeed, pleural tuberculosis is not a disease of childhood but of adult life. Of 194 cases observed by Osler,² only 10 per cent. occurred under twenty years of age. When pleurisy with effusion does occur in the child it differs in two respects from the condition as met with in later life. In the first place, consolidation instead of fluid may be thought of, because the respiratory murmur is so often tubular in character. More stress in diagnosis should be laid on the density of the dullness to percussion and the displacement of organs (mediastinum, heart, liver, and spleen).

The other direction in which pleurisy with effusion in the child differs from the disease in the adult is in the matter of

¹ L. Findlay, *Edin. Med. Journ.*, 1926, N.S., xxxiii., 147.

² W. Osler, *The Principles and Practice of Medicine*, London, 1905, 603.

prognosis. Subsequent development of pulmonary tuberculosis is what is generally feared, and it is estimated that in the adult this occurs in 30 to 40 per cent. of the cases. In the child, however, this sequel is exceptional. General tuberculosis or spinal caries are the usual sequelæ, but in the great majority of cases complete recovery takes place. Graham¹ found in R.H.S.C., Glasgow, that only 15 per cent. of the children developed any form of active tuberculosis afterwards, and Nobel and Steinebach² only found evidence of subsequent tuberculosis in at most 10 per cent. of the children with pleurisy under their care. In one example of pleurisy with effusion one of us (L. F.) observed the subsequent development of a localised cold abscess.³ Some writers⁴ describe tubercular empyema as a common sequela, but this has not been our experience.

Mention must be made of the condition which has been described within recent years as *epituberculosis*. The general conception is that this is an allergic reaction around a small tubercular focus, which gives rise to no constitutional symptoms, but which can be detected by physical examination and radiology. The lesion does not undergo softening with cavitation and recovers completely without leaving any trace behind. Its exact nature, however, is mere speculation, as there is no post-mortem evidence available. Tubercle bacilli are never detected in the sputum, so that there is always the possibility, in fact the strong probability, that the condition is not tuberculous at all. One has a difficulty in believing that a real tuberculosis, as suggested by Spence,⁵ could ever clear up. In our experience lesions conforming to the above description have been slowly resolving simple pneumonias which have come under observation after the crisis.

Tuberculous Skin Lesions.

The diseases of the skin due to tuberculosis include lupus vulgaris, scrofuloderma, and Bazin's disease, which are all caused by the local presence of the tubercle bacillus; and there are also eruptions, such as lichen scrofulosorum and

¹ S. Graham, *Glasg. Med. Journ.*, 1925, civ., 1.

² E. Nobel and R. Steinebach, *Zeit. f. Kinderh.*, 1922, xxxi., 98.

³ L. Findlay, *Rep. Roy. Soc. Med. (Sect. Dis. of Child.)*, 1932, xxv., 410.

⁴ C. A. Hedblom, *Surg. Obst. and Gynæcol.*, 1922, xxxiv., 445.

⁵ J. C. Spence, *Arch. Dis. Child.*, 1932, vii., 1.

other "toxi-tuberculides," which are set up by tuberculous toxin circulating in the blood.

Lupus is the commonest tuberculous skin affection, and generally begins in childhood. It is often due apparently to direct infection from kissing by a phthisical person, or from scratching with infected fingers; consequently it is frequently found on the face. When multiple patches appear after an attack of measles (Adamson) it may be due to the spread of the bacilli in the blood-stream from a breaking-down focus elsewhere in the body. The characteristic brownish-yellow, semi-transparent "apple-jelly" nodules are often easily seen; but sometimes their characters are masked by yellow crusts from pyogenic infection of the surface; and after a time the patch ulcerates.

Small patches of lupus are probably best treated by free excision, when this is practicable, or by the use of the sharp spoon or thermo-cautery. To larger areas, which cannot be so dealt with, an ointment of salicylic acid (10 gr. to 1 oz.) or oleate of mercury (5 gr. to 1 oz.) may be applied. In very chronic cases counter-irritation is sometimes beneficial. The treatment of severe and extensive lupus is, however, generally a matter for the dermatologist. The Finsen method, the X-rays, and radium have all given good results in skilled hands, but by far the best results are obtained with ultra-violet rays (Norman Walker).¹ Active general treatment of the tuberculous condition must not be neglected.

Lichen scrofulosorum is sometimes useful in drawing attention to the presence of obscure tuberculous lesions of the internal organs. Dr Cranston Low believes that its appearance is rather a good sign, for it is commoner in mild than in severe tuberculosis and is never seen in rapidly fatal and advancing cases. In addition to general treatment the skin should be soaked with cod-liver oil.

Mention has already been made of the *papulo-necrotic tuberculides* and their value in diagnosis (p. 959).

Tuberculosis of the Ear.

In school children aural tuberculosis is a rare disease, but in infants it is common in those places where the milk supply is contaminated by tubercle bacilli. Thus, Logan Turner and

¹ N. Walker, *Introduction to Dermatology*, 2nd ed., Edinburgh, 1932, p. 33.

J. S. Fraser¹ found that of the hospital cases of ear discharge under five years old examined in the Edinburgh Royal Infirmary, 9 per cent. were tuberculous; while, among those under twelve months, no fewer than 50 per cent. were due to this cause.

The disease begins in the tympanum; and it may get there either by direct spread along the mucous membrane from the naso-pharynx, or by the bacilli being carried to it through the lumen of the infant's short and wide Eustachian tube. It is probable that primary infection of the temporal bone through the blood-stream rarely, if ever, occurs. The middle ear, when once infected, becomes rapidly disorganised; the drum is destroyed, the ossicles exfoliate, and the petrous and mastoid bones become necrosed; the facial nerve is often also implicated. At the same time the periotic glands almost always become enlarged and caseous, and often break down. Occasionally the disease extends directly to the meninges.

In the large majority of cases the subjective symptoms are few and trivial, and neither pain, restlessness, nor fever is noticed; so that the first thing to draw attention to the ear is the profuse discharge of thin curdy pus from the meatus, with enlargement of glands and perhaps facial palsy.

The *treatment* consists in early opening of the mastoid antrum, the removal of sequestra, and the establishment of free drainage from the affected cavities, along with removal of caseous glands. The operation must be followed by vigorous general treatment; and a vaccine for pyogenic organisms is sometimes also beneficial.

The prognosis is not favourable. The operation, though occasionally successful, often fails to eradicate the disease; and the risk of its spread to the meninges is considerable.

¹ Logan Turner and J. S. Fraser, "Tuberculosis of the Middle-Ear Cleft in Children," *Journ. of Laryngology, Rhinology, and Otology*, June 1915, 209.

CHAPTER XXXVIII

NURSERY HYGIENE

Clothing

THE details—even the minute details—of a child's clothing are worthy of attention. The main principles which should regulate them must therefore be briefly referred to.

General Principles. — A child's clothing should be uniformly warm, soft, and light. It should also be moderately loose, so that it does not exert undue pressure on any part of the body and can be quickly put on and off.

The clothes must be sufficiently **warm** not only because this is necessary in order to prevent chills, but because, if the body is not adequately protected, it has to waste more nerve energy in heat production than it can fairly afford. Children's heat-regulating powers are feebler than those of adults; sufficient clothing is therefore especially important for them. Any attempt at hardening a child, as it is called, by underclothing him, is to be regarded as even more foolish than overheating him by too many or too thick clothes.

It is also extremely important that, in little children, the protection from cold provided by the clothing should be uniform. Many recurrent catarrhs of the respiratory and digestive tracts, in cold weather, are due to bare arms and legs and scantily covered bodies. Long sleeves and stockings and a knitted or flannel binder are very effective in warding-off such attacks.

When a child exposes himself to the risk of chills by constantly kicking off the bed-clothes it is a good plan to give him a long bedgown, the lower end of which is fitted with a string like the mouth of a bag, so that when it is drawn tight he cannot get his legs out.

Paralysed limbs, especially, should be well covered, both in the daytime and at night. Warmth favours their circulation and probably lessens the often inevitable atrophy,

Should ordinary thick or double stockings or sleeves not be sufficient, it is well to have them padded with cotton-wool.

The baby's clothes should be **soft, light, and loose**, because his skin is soft and easily chafed, and his movements are readily hampered by slight restraint. Constriction of any part of the body or limbs is bad for the normal growth and development.

Lastly, little children ought to have their dress so arranged that it can be **put on and off** as **quickly** as possible. This is very important in the case of feeble and sick infants, who may suffer seriously if they are wearied, irritated, and chilled by an unnecessarily complicated process of dressing and undressing. In all serious illnesses in little children it is a good plan to discard ordinary clothes with their numerous buttons, tapes, and bands, and to use instead simple flannel night-clothes lined with cotton-wool.

When we come to apply these elementary principles we find that woollen garments of loose texture (*e.g.*, knitted ones) are the best for a child to wear, especially next the skin. Wool is a bad conductor of heat, but is easily permeable, so that it does not interfere with the free evaporation of moisture from the skin. The only exception to this is in the case of the napkins which babies have to wear until they gain some measure of control over the bladder. These are generally made of linen or cotton, because they have to be frequently washed. Occasionally we find a mackintosh worn over the napkin. This is a useful measure under special circumstances, as on a journey, but when used habitually, merely to save trouble, it is most objectionable, as it is sure to cause irritation of the skin. When soda, or strongly alkaline soap, is used in washing the napkins there is also a risk of skin irritation (p. 427).

When, in cases of diarrhœa, there is much redness of the parts surrounding the anus caused by irritating fæces, it is sometimes a good plan to place a large pad of absorbent cotton-wool inside the diaper. This absorbs the liquid fæces in such a way as to prevent their coming in contact with so large an area of skin as is the case when the motion spreads itself out over the napkin; and the pad of wool can be frequently changed.

The child's bed-clothes should not be so thick and heavy as to overheat him, but they require to be a little warmer than

those he wears during the day, to make up for the want of exercise while he is sleeping.

Fresh Air and Sunshine.

Fresh pure air, cleanliness, and warmth are absolutely necessary for perfect health, and sunshine is especially important. Children, like flowers, get pale and droop without the sun. The nursery should therefore be the sunniest, driest, freshest room in the house. It should have a southern exposure, be well ventilated, and kept at a temperature of 60° to 65° F. The windows should be widely opened several times during the day to air the room thoroughly, and one window at least should be constantly open for some inches at the top.

The children of many of the respectable poor are less robust than those of their more careless neighbours, just because of the care with which they are protected from cold and at the same time from fresh air. The close atmosphere of unventilated rooms is especially dangerous during convalescence from such diseases as measles and whooping-cough, and may make all the difference between complete recovery and the development of tuberculosis.

Draughts.—While seeing carefully to the ventilation, however, we must, in some forms of illness, be careful to avoid draughts. For healthy children the risk of injury from draughts is much less than that from too little fresh air; but for those who are liable to recurrent bronchitis or rheumatism the danger is a real and important one. It is well to remember that in cold weather large plate-glass windows cause a constant draught in their immediate vicinity. Double windows or double panes, by forming an air cushion, are a protection against this effect.

Going out.—As a general rule a baby should be taken out every fine day, summer and winter. If he is born in summer he may go out for the first time about a week after birth; if in winter, this may require to be delayed for a month, but it entirely depends on the weather. So long as there is fog and the winds are cold the child should be kept indoors. At first he should not be out longer than from fifteen to twenty minutes, but later, if the weather is warm and dry, he can scarcely be too much in the open air. It is important not

to allow young or delicate children to be exposed to windy weather, even although the thermometer is not very low, because they are readily chilled by wind. In the same way it is important even in this country that babies should be carefully protected from the sun in warm weather. The evaporation which takes place from wet roads is often a source of chill in the case of delicate children who are old enough to walk, even when the weather overhead is not very cold.

Little children should not be taken out, even in good weather, if they are coughing and sneezing or otherwise suffering from the results of a recent chill. When, however, children who are past babyhood are in good health, it is safest in the long run to err, if at all, in sending them out too much or in too cold weather, rather than in keeping them too much shut up. If a child has a tendency to eczema he should be carefully protected from cold winds.

The great value of open air in the treatment of bronchopneumonia has already been referred to (p. 627).

Washing and Bathing.

A baby should have one bath every day, and if he is strong he may have two. Harm is certainly often done by bathing delicate children too much. For babies who are not strong one regular bath, and sponging in place of a second, is much better than two baths.

The temperature of the bath should be about 90° F. in the case of young babies, and if they are delicate it is always well to use a bath thermometer. It should never be warmer than 95° F. As the baby grows older the bath, especially in warm weather, may be reduced to 85° or 80° F.

Very little soap is required for the general surface of the body in a young infant, and soaps containing much free alkali are to be avoided. For children with delicate skins some form of overfatty soap is best; but for ordinary healthy children plain unscented soap does quite well.

The bath should be given in a part of the room where there is no draught, and the baby should not be in it more than five minutes. The drying process after it must be rapid and thorough. It is customary after drying the baby to apply some soothing powder to those parts where

folds of skin are in apposition. For this, oxide of zinc, starch, and boracic powder may be used. Should there be any tendency to intertrigo some simple ointment such as zinc ointment, cold cream, or vaseline is better.

If a baby's extremities get very cold after the bath, and it seems to weaken and depress him, it will be well to stop it and only to have him sponged instead.

The Cold Douche.—Ordinary cold douches are very good for many older children, but for the little ones in the nursery, and those who are delicate among the older children, they should only be given in modified forms and always with caution and attention to details.

The best way to give a cold douche to little children is, after they have been washed in warm water and are still sitting in it, to pour some cold water from a big sponge or from a jug over their shoulders and to take them out at once and dry them thoroughly.

When a cold douche is to be given to a delicate child, he should always stand in a little hot water while he is having it. When this precaution is taken the bath will often do good when otherwise it would not have done so. Care should also be taken that the room is warm and that the child is not allowed to chill himself before the bath begins by hanging about or playing without sufficient clothes on. *Disregard of precautions in such matters often results in recurrent ailments which are attributed to special delicacy.* It is a good plan to have the child shampooed thoroughly all over for five or ten minutes before the bath, and energetic friction with a rough towel after it is stimulating and beneficial.

Uses of the Cold Douche.—Cold douching carefully carried out is extremely useful for many children. It stimulates the nervous system, improves the circulation, trains the heat-regulating mechanism, and often does away with the coldness of the feet which is such a troublesome symptom. The appetite also increases under its use, and the children become altogether healthier and happier and are less liable to take cold.

Modification of Cold Douche.—Sometimes the cold douche disagrees with the child and causes unpleasant symptoms. For example, instead of its leaving him with a healthy glow and an increased appetite and generally brightened up, he may be pale and shivery after it, with cold blue fingers and a disinclination

for food ; or the healthy reaction may only last a short time and be followed by weariness, headache, and a feeling of chilliness.

If a cold bath, given with all due care and precaution, results in either of these conditions, it must be modified or discontinued. Often a tepid salt-water bath does very well in such cases, the salt increasing the stimulating effect of the water on the skin. Again, in children with a weak circulation, thorough rubbing of the body with a coarse towel which has been wrung out of cold water is an excellent substitute for a regular bath.

In other cases the cold bathing may be limited to certain parts. Children with cold feet who cannot stand an ordinary douche may be benefited by having their feet bathed in cold water and afterwards briskly rubbed, and a similar cold sponging of the throat and shoulders is useful for diminishing the liability which some children have to take throat colds. Lastly, many children who cannot stand cold bathing in winter may benefit from it greatly during the summer months.

Hot Baths.—While cold baths may do harm sometimes, hot baths are just as likely to do so if injudiciously given. They must not be given too hot or too often (once in the week is enough), and the child should not be long in them—never more than ten minutes at most. The effect of such a hot bath is stimulating, but a long-continued one is weakening and relaxing. It is important that the child should not catch cold after the bath ; hence it is better to give it just before he goes to bed.

Sea-water Baths.—Sea-water baths are good for children of all ages, but a child should not usually be allowed to bathe in the sea until he is six or eight years old, or oftener than once a day. The best time for a sea-bath is three hours after a meal, but the child may go before breakfast, provided he has a biscuit and milk before starting. The bath should never be taken immediately after a meal, and never when the child is feeling chilly or is hot and perspiring. The head should be wetted first, and the child should not, even in hot weather, be allowed to stay in longer than fifteen or twenty minutes. After the bath he should be rapidly dried and dressed, and should have a sharp walk and a biscuit or a glass of milk.

Sea-bathing should make a child hungrier and in better spirits. If he gets dull and chilly and seems out of sorts while he is having it, this indicates that it is not agreeing with him

and that it should be stopped. Children who have perforation of the tympanum should not be allowed to bathe in the sea.

The Hair.—The hair must, of course, be kept clean, but much washing, especially with soap, certainly causes dryness. In little boys with short hair the head should be washed every day ; but in older children with long hair once a week is enough. If, with an ordinary amount of washing, the hair gets very dry, it may be necessary to use a small amount of oily matter of some kind to replace the natural grease which washing has removed ; but, as a general rule, it is better in every way to put no pomade of any kind on the hair.

If scurf gathers on the scalp it should be removed by washing with soap and water or by spirit of soap. A comb should not be used for this purpose, as it is apt to injure the roots of the hair. For the same reason the brushes used for children's hair should be soft, and the teeth of the comb for ordinary use should be blunt and not too close together.

CHAPTER XXXIX

THERAPEUTICS

Baths and other External Applications for Therapeutic Purposes

Cold or Tepid Sponging.—Cold sponging is useful for reducing temperature in children, and should generally be tried before having recourse to stronger measures. The water may be used at 80° F., and the process should last five or ten minutes. Care must of course be taken that the child is not unnecessarily exposed nor his bed wetted.

The Wet Pack.—The ordinary wet pack is also of value, being easily applied and more efficacious in reducing temperature than simple cold sponging. A small sheet or large towel wrung out of cold water is rapidly folded round the child, and he is then wrapped up in a blanket. The pack may be repeated in twenty minutes unless the child is sleeping, in which case he may be left in it for an hour or more. Another method of applying the wet pack is to wrap the child in the sheet or towel wrung out of tepid or warm water and gradually reduce its temperature by pouring on or squeezing out of a large sponge water of gradually lower temperatures. During the process the child's temperature must be carefully watched and the child removed when the temperature has reached 100° F., as it will continue to fall for some time afterwards.

Local wet packs or wet compresses may be used in many illnesses. The application of a wet stocking round the neck, for example, is an excellent domestic remedy for a simple sore throat; and a wet compress around the abdomen at night is soothing and efficacious in many cases of indigestion with discomfort.

The Mustard Pack.—This often acts well as an external stimulant for cases of collapse or prostration; and causes less disturbance to the patient than the mustard bath. A table-

spoonful of mustard is mixed with a quart of tepid water, and a towel is dipped in this and swathed round the whole body. The pack may be continued for ten or fifteen minutes, at the end of which time the body will be distinctly red. Similar applications to the chest are often useful in cases of bronchitis and collapse. During the intervals between their application a cotton-wool jacket should be worn.

The Cold Douche.—The best method of applying the cold douche, and its stimulant value for delicate as well as for strong children, have been already discussed (p. 989).

The Cold or Tepid Bath.—This is a useful means of reducing temperature in children, and their small size makes it easy of application. The patient should be put into water at about 100° F., which is then gradually cooled to about 80°, or even 75°, by adding cold water or ice. The child may be kept in the bath from five to fifteen minutes, but the state of the temperature and pulse must be watched all the time.

The Warm Bath.—The warm bath (90° F.) is a soothing application in cases of convulsions and laryngeal spasm. It acts also as a diaphoretic, and may be given in the early stage of measles and other eruptive fevers to encourage the rash to come out more fully. The child may be kept in it from ten to twenty minutes.

The Hot Bath.—A hot bath (100° F.) is of great value as a stimulant, especially in children exhausted by severe diarrhoea and vomiting, and in those with pulmonary collapse. The child must not, however, be allowed to remain too long in the water or he will be depressed. For an infant, three minutes are long enough, and for an older child five or ten. The child should be rapidly dried after the bath and put between blankets, with a hot bottle at his feet.

The Mustard Bath.—The addition of mustard to the hot bath makes it more stimulating. One ounce of mustard may be used in each gallon of hot water. The mustard is made into a paste with a little cold water and then gradually stirred into the bath, or it may be put into it in a muslin bag. The child is held in the bath till the arms of the person holding him begin to tingle. This is a useful remedy in cases of prostration and collapse of any kind—especially in young babies.

Ice-Bags.—These must be used with great care in infants, owing to the danger of depressing them too much. If the

child is restless there may be difficulty in keeping the ice-bag properly applied.

An ice-bag is frequently used in acute head cases, and great benefit may sometimes be derived from it in acute pericarditis (p. 594).

Hot Fomentations.—These are made with flannel or spongio-piline. They have the advantage that they are cleaner than poultices and require less skill for their proper application.

Poultices.—Poultices are not so much used now as they formerly were. This is partly because it is so difficult to get them properly made and applied; and their place is largely taken by hot fomentations. In many cases, however, where a child is suffering from uneasiness in the abdomen from any cause, a large poultice is very soothing, and may secure a measure of relief which it would be difficult to obtain in any other way. Poultices are also very useful over the chest when there is pleuritic pain, and across the loins in nephritis. The temperature of the poultice must not be too high or the child may be severely burned. A poultice which the nurse cannot bear against her own cheek should not be applied to a child. The danger of burning is much reduced by oiling the skin well beforehand.

Mustard Poultices.—Mustard poultices are valuable in many cases. For young babies they should be made with one part of mustard to five of linseed meal. The mustard and linseed are mixed together dry in a bowl, hot (not boiling) water is then added, and the poultice is made, and applied on a handkerchief. It may be left on for six or eight hours, and should be followed by a thick layer of cotton wool. For older children the proportions may be one to three or four, but the poultice should not be left on longer than four hours (Appendix E, Form. 63 and 64).

Pure mustard must never be applied to a baby's skin, as it is apt to produce serious sloughing. In older children a small mustard plaster or a piece of mustard leaf the size of a penny may often be applied to the episternal notch with great effect in cases of irritating throat coughs, such as are met with in chronic tracheitis, and when the eruption is beginning in measles.

Other Ways of Applying Heat.—Hot bottles are, of course, of great value in many cases where the child's vitality

is lowered or his extremities chilly. A simple and useful method of applying warmth to an infant's cot by means of electric light is shown in Fig. 344.

Blisters.—In young children blisters should scarcely ever be applied, and even in older children they must only be used with great care, on account of their tendency to cause sloughing and the risk of their irritating the kidneys. Their effect varies in different children and it is generally best, to begin with, to leave the blister on for one or two hours only, and if it has not risen then, to apply a poultice over the place. Blisters are sometimes useful in pericarditis with effusion

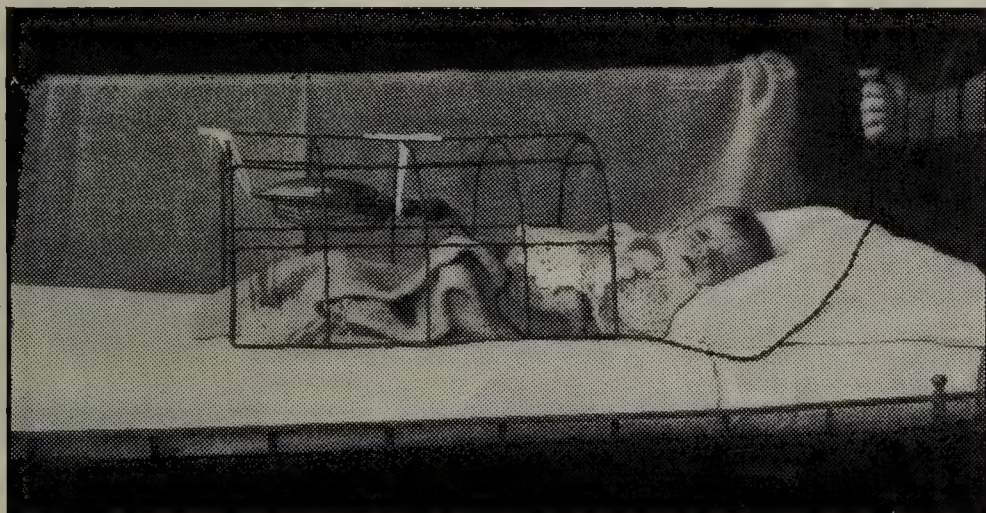


FIG. 344.—Simple method of warming bed by electricity.

and in endocarditis; also in some cerebral cases where there is an increase of fluid in the ventricles.

Bleeding.—It is generally accepted that in old times the practice of blood-letting was carried much too far, and that in children injudicious venesection or leeching is more dangerous than in adults. Certainly, however, in recent years many have gone too far in the opposite direction and have abstained from bleeding when it would have done a great deal of good. Our own experience has been entirely in accordance with that of Dr Lees, that in suitable circumstances bleeding “is a remedy of priceless value, capable of giving immediate relief impossible by any other means.”¹

The condition which most frequently calls for blood-letting is rapid over-distension of the right heart such as occurs in acute pericarditis and nephritis, in the later stages of mitral

¹ D. B. Lees, *The Treatment of some Acute Visceral Inflammations*, London, 1904, 30.

stenosis, in acute pneumonia, and during acute exacerbations in chronic bronchitis. The chief indications are severe dyspnœa and the physical signs of a distended right auricle (p. 579). Bleeding is also of value in uræmia, and in acute meningitis and otitis in strong children.

In older children venesection is in some ways preferable, but in infants the operation is often extremely difficult, and if *leeches* can be got they are much more convenient. The best place for their application, in cases of distended right auricle, is below the right nipple. In this situation they do not interfere with the subsequent examination of the heart, and the bleeding can be arrested by pressure against the ribs. In bad cases two leeches may be applied in the case of a baby and from three to six in that of an older child.

Blood may also be taken in infants, from the superficial veins on the scalp or elsewhere, by means of a syringe and needle.

Dry cupping is sometimes useful, especially in renal cases when uræmia threatens.

Medicinal Treatment.

In the treatment of children there can be no doubt that drugs should, in most cases, occupy an altogether subordinate place. Generally, directions as to such matters as food and drink, fresh air and exercise, clothing and rest in bed, freedom from causes of worry, and pleasant occupations, are far more valuable than any medicines. In practice these things are too apt to be taken for granted when medicine is prescribed. Not only should definite directions be given about them, but when these are important they should be written down. Unless this is done the details on which their efficacy largely depends are often forgotten or misunderstood.

While the value of drugs, however, in the treatment of children has sometimes been exaggerated, it is often also altogether under-estimated. In suitable cases many medicines are of the greatest value.

Dosage.—Although posological tables may sometimes help the beginner, their usefulness is limited. In fixing the dose, the child's condition as to size, weight, and strength is often quite as important as his age. We have also to remember that while some drugs, such as opium, have to be given with special

caution, others, like digitalis, mercury, and quinine, are well borne, and one or two, such as belladonna and arsenic, can usually be taken in larger doses by children of five to ten years than by adults.

It is particularly important to have fluid medicines measured, if possible, in a measure-glass rather than in a teaspoon or by drops. The teaspoons in ordinary use differ considerably in size, and many of them contain at least 90 minims. The size of drops also varies, not only with the nature of the fluid measured, but also with the shape of the bottle from which they are dropped.

The medicine should, of course, be made as palatable as possible, and a large dose should not be ordered if a small one will do as well. When obstinate refusal of medicine occurs in grave illness it is a question whether the consequent struggle may not do more harm than the medicine does good.

A few remarks may be made about some of the drugs which are most useful in the treatment of children.

Alcohol.—Stimulants should never, of course, form part of a healthy child's diet, but in debility great benefit sometimes results from small doses of wine or beer or diluted brandy taken with food. Alcohol is also occasionally of great use in acute disease in childhood, as in later life. In ordinary feverish illnesses it is not required; but in some cases of heart failure, or if a typhoid condition should set in, it may be urgently called for. It is also useful in exhausting conditions, such as the pulmonary and other complications of whooping-cough or measles, and in septicæmia.

Alcohol may be given in the form of brandy, whisky, wine, or sherry whey. Champagne is sometimes retained when everything else is vomited. When whisky or brandy is ordered for young babies it must not be given too concentrated—not more than 10 or 15 drops to the teaspoonful; and it is always well to indicate the minimum to be given in the twenty-four hours, and the amount that is not to be exceeded. A baby of a year or two old may have from 2 drm. to $\frac{1}{2}$ oz. of whisky or brandy in the twenty-four hours, and this may be increased in a few cases to 1 or even $1\frac{1}{2}$ oz. To a child of three or four double this amount may be given. Small doses frequently repeated and well diluted are preferable to larger doses given at longer intervals.

Sherry whey (see Appendix F) forms a useful way of giving alcohol to young babies. The nourishment it contains, though not large in amount, is in a very digestible form. The following analysis is given by Myers and Still¹:—

Protein (mostly lactalbumin)	. . .	0.45 per cent.
Fat	0.95 „
Milk sugar	5.00 „
Alcohol	2.30 „

It has a decidedly acid reaction. The alcoholic strength when made from “cooking sherry” is about one-eighteenth that of brandy. If good sherry is used, a larger amount of it is necessary to produce coagulation and the alcoholic strength is therefore greater. Sherry whey is often retained by an irritable stomach. When there is flatulence and colic it has often a markedly carminative effect.

Tonics.—Tonics are sometimes of great value, and their use will often speedily bring a child who has been ailing back to his normal condition. The use of iron preparations, however, is much restricted owing to their often upsetting the digestion.

Cod-liver Oil.—Perhaps the best and most generally useful of all tonics for young children is cod-liver oil, but it should not be ordered if there are signs of dyspepsia; and if its administration interferes with the child's appetite or digestion it must be discontinued and an alkaline or acid tonic substituted. This often improves the digestion so much that the child is able to take the oil with benefit. Those who can take cod-liver oil well in winter are sometimes unable to digest it during hot weather. From 10 to 30 minims thrice daily is a sufficient dose for most young children, and it may either be given pure or in the form of an emulsion. If it agrees well the dose may be increased to a teaspoonful; but if undigested oil is noticed in the motions the amount should be diminished.

Iron.—Iron is a most useful drug in children. It has, however, to be given with caution because of the risk of its disagreeing with the digestion. A convenient form for its administration is reduced iron. Of this $\frac{1}{2}$ to 1 gr. may be given to infants thrice daily, and 1 or 2 gr. to older children. It is readily taken, and has the advantage over liquid preparations that it does not discolour the teeth. When constipation is

¹ Myers and Still, *Lancet*, 12th Jan. 1907.

present the sulphate may be given along with sulphate of magnesia (Appendix E, Form. 24).

Arsenic.—Arsenic is usually given in the form of Fowler's solution. It may be given, as a tonic, in doses of 2 to 5 drops well diluted thrice daily after food. It is also recommended in much larger doses (15 minims t.i.d.) for a short time in chorea. It is also useful in minute doses in lenteric diarrhœa and allied conditions (p. 313).

Quinine.—Quinine is one of the drugs which children bear well and in comparatively large doses. It is usually given in solution, 1 gr. of the sulphate to a teaspoonful, and some flavouring or sweetening substance (such as syrup of orange-peel 20 minims to each $\frac{1}{2}$ gr.) may be added, though none of them is very successful in disguising the taste. Larger doses of the powder may be shaken up rapidly with syrup and swallowed before much has been dissolved. Probably the best way to administer it to young children is to order it suspended in glycerine (1 gr. to the drachm) and to direct the nurse to give the dose in a wineglassful of milk (E. Smith). In children over three the sulphate may be given in the form of capsules, or it may be made up into very small pilules which can be mixed with jelly and thus swallowed (West). The tannate may also be given in powder or with chocolate. It is comparatively tasteless, but has to be given in three times the quantity of the sulphate, and is said not to be so reliable in its action. If quinine cannot be given by the mouth, it may be administered in the form of suppositories or as an enema—double the ordinary dose being used. It may also be given hypodermically, but this method is rarely warranted in children, except in severe malaria.

Strychnine.—Solution of strychnine and tincture of nuxvomica are useful tonics, and are usually taken quite well if sufficiently diluted. Of the former, $\frac{1}{3}$ to 1 minim thrice daily may be given to children of one to five years old, and 2 minims to older children. Of the latter, twice these doses may be employed.

Digitalis and Strophanthus.—These are well borne, in suitable cases, even by young children.

Mineral Acids.—The dilute acids are often of benefit in dyspepsia; and they are useful in diarrhœa, on account of their anti-fermentative action. One or 2 minims of dilute hydrochloric

or nitric acid may be given in each dose to infants of from six to twelve months, and from 2 to 4 minims to children of from five to ten years. It is well to dilute the acid to the extent of a teaspoonful to each minim, otherwise there may be difficulty in getting a child to take the medicine on account of its taste.

Alkalies.—There are few medicines which are so often successful in temporarily improving the condition of weakly children as alkaline tonics. Alkalies are also of great value in the treatment of many nervous and other ailments arising from an acid fermentation of food in the alimentary canal, in acetonæmic conditions, and in *Bacillus coli* infections of the urinary tract.

Mercury.—Grey powder, calomel, and solution of perchloride of mercury are much used in childhood, not only in syphilis but also in various dyspeptic disorders, on account of their anti-fermentative action. Calomel is a useful purgative in many conditions. One gr. of it may be given to a child under twelve months, 2 gr. to one of two years, and 3 gr. to those between three and seven years old; or $\frac{1}{6}$ gr. may be given every hour until the bowels act. In cases of acute tonsillitis and commencing laryngeal catarrh the treatment should generally begin with a mercurial purge. The value of mercury as an adjuvant to digitalis in some cardiac cases has been already referred to (p. 592).

Weak mercurial ointments (*e.g.*, nitrate or ammonio-chloride of mercury, 5 gr. to 1 oz.) are very useful in the treatment of impetiginous eczema and of most forms of superficial sore in strumous children.

Opium.—To premature babies, and those who are suffering from severe or acute respiratory distress, opium should never be given; but, in many circumstances in childhood, it is a most valuable medicine.

It is chiefly of use in relieving pain and in quieting the action of the bowels. It is useful in some forms of diarrhœa because, by diminishing the rapidity with which the other remedies pass through the bowel, it gives them time to act. It is also sometimes useful in allaying spasm, as in whooping-cough and croup, and in soothing irritable coughs, but it should never be used for ordinary restlessness or to procure sleep. The value of opium during the onset of croupous pneumonia has already been referred to (p. 621).

The dose must be regulated by the size and strength of the child as well as by his age—a wasted baby of a year old requiring a smaller dose than a strong child of six months. Some children are specially intolerant, and some more than usually tolerant of the drug. Cases of extreme susceptibility to opium in strong children are very rare indeed, and if reasonable precautions are taken no danger of poisoning need be feared. The effect of the first dose should always, however, be watched; and if the child is not to be seen again for some time it is well to instruct the mother not to repeat the medicine if he is drowsy, and to stop it whenever the symptoms for which it is being given are relieved.

Laudanum, nepenthe, and solution of hydrochlorate of morphine (B.P.) may be taken safely by children a year old in doses of $\frac{1}{2}$ to 1 drop, while to those of six months, $\frac{1}{6}$ to $\frac{1}{3}$, and to those of two months, $\frac{1}{20}$ of a drop may be given to begin with. A child of twelve months may have $\frac{1}{2}$ to 1 gr. of Dover's powder.

Tinct. opii camph. is a convenient form in which to give small doses of an opiate to children. To infants during the first year, 1 drop for each month of their age, and to a child of five years, $\frac{1}{2}$ drm. may be given.

Codeine is sometimes helpful in cases of abdominal pain and tenesmus, and for coughs. For a child of a year old, $\frac{1}{20}$ of a grain is a suitable dose and from $\frac{1}{12}$ to $\frac{1}{6}$ of a grain for one of four or five years. Syrup of codeia, which contains 2 grains to the ounce, is a safe and useful method of presenting this drug. Half-drachm doses in babies and drachm doses in older children can be given. Heroin ($\frac{1}{100}$ to $\frac{1}{30}$ of a grain) is sometimes useful in persistent coughs. Morphine may be given hypodermically to a strong infant of a year old, in doses of $\frac{1}{30}$ to $\frac{1}{24}$ of a grain.

Chloral Hydrate.—Chloral is well borne by children. It is especially useful in cases of infantile convulsions (pp. 671 and 673) and in those which complicate whooping-cough. In young infants it is best given per rectum (1 gr. for a child of a month, 5 gr. for one of six months, and 10 gr. for one of a year old). It may be given by the mouth to children of one or two years old in doses of $2\frac{1}{2}$ to 5 gr., and to older children in doses of 5 to 10 gr.

Bromides of Potash, Soda, and Ammonia.—The bromides are useful in the irritability of teething, in sleeplessness, and

in nervousness of any kind. If a nervous child has to undergo a slight operation, without chloroform, it is sometimes an advantage to give him a large dose of bromide. Although this will not diminish the pain of the operation, it will make the patient drowsy and lower his sensibility, so that he will suffer less from the fright of it than he would otherwise do. In a similar way, children who are made sick by railway travelling are sometimes greatly benefited by a dose of bromide before they start.

Under ordinary circumstances, 1 or 2 gr. every two or three hours may be given to infants of a month or two old, while children of a year or more may have 3 to 5 gr. repeated at similar intervals.

When bromides are being given for any length of time it is well to combine with them a small dose of liquor arsenicalis to lessen the risk of acne.

Antipyrine is a very useful drug in childhood under a variety of circumstances. Its main value depends on its sedative action. To children who are atrophied or prostrate from acute disease it must be given with caution, if at all. Generally, however, it is well borne even by young infants.

It is often valuable in cases of whooping-cough which are not complicated by much bronchitis, and also in chorea. For night-terrors and restlessness, and for laryngismus and spasmodic croup, it is a more trustworthy sedative than the bromides. It is also useful in colic and in the pains accompanying dentition in young infants. In cases of high temperature from septicæmia and pneumonia it often relieves the restlessness even when it has little or no effect on the pyrexia. One gr. every four hours may be given for each year of the child's age during the first three years. For night-terrors, in a child of from five to ten years, a dose of from 5 to 10 gr. may be given.

Belladonna is a drug which children bear remarkably well. The tincture may be given to babies of a year in doses of 2 or 3 minims, and for children of six or eight years from 10 to 20 minims is a usual dose. It is useful in some cases of broncho-pneumonia, pulmonary collapse, and whooping-cough, and especially in enuresis.

Thyroid Substance.—Thyroid is usually most conveniently administered in the form of Burroughs and Wellcome's tabloids. It is sometimes useful in other conditions than myxoedema

and cretinism. It may, for example, be given with advantage in some cases of obesity and in enuresis.

Emetics.—In acute indigestion, emetics are valuable in getting rid speedily of the contents of the stomach. In bronchitis also they are of great use in helping to clear out of the bronchi any secretion which may be blocking them and which is not effectually removed by coughing.

The most effectual and safest emetic is powdered ipecacuanha, and it should be given in 5-gr. doses every ten minutes until vomiting occurs.

Ipecacuanha wine acts well when it is fresh, but its emetic properties are greatly diminished by keeping. We may also give sulphate of copper ($\frac{1}{2}$ gr. every ten minutes) or alum (1 dr. in syrup at similar intervals). Apomorphine should not be used, as it is too depressing.

Purgatives.—Castor oil is generally regarded as the safest and most reliable of purgatives. Castor oil should never be prescribed for constipation, but only as a purgative. Its taste, which is its chief disadvantage, may be disguised to a considerable extent if the dose is shaken up in a bottle with a wineglassful of hot milk, sweetened and flavoured by a piece of cinnamon having been boiled in it. Another successful device is to give the child a mouthful of dry oatmeal immediately before he takes the oil.

Senna may be given in the form of the syrup of senna pods, or as compound liquorice powder. These preparations are easily taken, but they are more apt to cause griping than castor oil. They must always be used fresh.

Rhubarb may be given to older children in cachets, and for younger children the syrup is a convenient form.

Carlsbad salts (a teaspoonful) is a useful form of saline purge for older children, and a tablespoonful of any of the aperient waters may be given with an equal quantity of hot water or hot milk.

Hypodermic Injection of Medicine.—Subcutaneous injections are extremely useful in certain serious and acute conditions. For example, morphine may be injected in convulsions and cholera infantum, adrenalin in cases of asthma, and ether and strychnine in states of collapse.

Hypodermic Injection of Saline Solution.—The injection of sterilised salt solution into the subcutaneous tissue is often

of the greatest value in cases where much fluid has been removed from the body by continued vomiting or diarrhœa; and also when a similar drying-up of the tissues has resulted from the child having refused for any reason to take the breast or bottle.

The necessary apparatus for giving such injections consists in a needle, about the size used for exploring the chest, 2 feet or so of tubing, and, for a funnel, the outer part of a 2-oz. glass syringe. The fluid used is a $\frac{3}{4}$ -per cent. solution of sodium chloride, and it, as well as the apparatus, must of course be sterilised by boiling.

The fluid should be considerably above the body temperature to begin with, because it is rapidly cooled in the funnel and tube. When filled, the funnel should be covered completely with cotton wool, and part of the tube may be allowed to lie in a basin of hot water. The needle may be inserted into the subcutaneous tissue in any convenient part of the body. The back, pectoral regions, abdominal wall, and thighs are the best places. From 1 to 6 oz. of fluid may be used at a time. Generally from 2 to 3 oz. are sufficient. The funnel is suspended about a foot above the patient and the solution is allowed to flow in very slowly. In most cases no pain whatever is caused after the needle is in place. In some children, however, the distension of the tissues seems to hurt a good deal. The skin should be carefully covered with cotton wool during the injection, as it is apt to get very cold. The puncture is closed with collodion.

Applications of Drugs to the Skin in the form of fomentations and compresses are to be used with great caution in childhood. Carbolic acid is especially dangerous because of the ease with which it is absorbed through the skin and from raw surfaces. It is probable that, in some of the cases of burns which end fatally, death is due largely to the absorption of antiseptic drugs from the surface. Methyl salicylate or oil of wintergreen, if applied too liberally, may cause a fatal acidosis.

Applications to the Throat.—Morbid conditions of the pharynx and tonsils are generally most satisfactorily treated by the application, with a large brush, of some bland application which will do no harm if it is swallowed. In some children a spray may be used, but in most the brush is more effectual. Gargling should not be ordered for children under seven years,

and, if it is prescribed at all, care must be taken to make sure that the child understands how to do it.

Applications to the Nose and Naso-Pharynx.—There are various ways of applying alkaline and antiseptic lotions to the nasal cavities and through them to the naso-pharynx. The lotion may be sniffed up by the child from his own palm or gently poured into the nostrils from a spoon, the head being tilted back slightly. The best way, however, in young infants is to drop the liquid into the nose by means of an ordinary medicine dropper, while the child is lying on his back.

The Mechanical Treatment of the Stomach.

The mechanical treatment of the stomach has a wider application in young children than in adults. It may be considered under two heads: 1. Forced feeding, or gavage; 2. Stomach-washing, or lavage. Both these measures are simple of application and of great value in suitable cases.

1. Forced Feeding, or Gavage.

Methods and Apparatus.—When a child is unable or unwilling to swallow he may be forcibly fed either by the nose or by the mouth.

(a) *Nasal Feeding.*—The simplest form of nasal feeding consists in pouring a bland form of liquid nourishment into one nostril, through which it rapidly finds its way to the pharynx, and is inevitably swallowed. The child must be kept lying on his back and his head held steady. The food given must of course be quite unirritating in character (*e.g.*, sterilised milk). The process of feeding must take place slowly, and regular intervals must be allowed for swallowing. Should the food be of a kind which might irritate the delicate nasal mucous membrane, a tube must be used long enough to reach to the back of the pharynx, if not into the œsophagus. In most cases, however, it is advisable to do nasal feeding by means of a catheter which is passed into the œsophagus.

For this a soft rubber catheter (No. 4 or 5) is suitable. It is thoroughly lubricated, and passed into the nostril with the patient lying on his back, or in older children while he is sitting up. When the end of the catheter reaches the pharynx

there is often retching, and some resistance is felt. The patient's head should then be inclined slightly forward, and the tube pushed gently on. As it gains the œsophagus it generally ceases to irritate the pharynx, and soon the passage of gas and liquid from its upper end indicate that it has reached the stomach.

The catheter may pass into the larynx, but this cannot happen unless the swallowing reflex is abolished; its occurrence is announced by coughing and dyspnœa. More frequently the catheter passes into the mouth, and this is likely to occur if there is much coughing and retching while the end of the catheter is passing the pharynx. When the catheter has reached the stomach and the retching has stopped, the food is introduced into it through a funnel.

The best funnel for this, and for any similar purpose in children, is formed by the barrel of a glass syringe with a fairly wide nozzle. When the catheter is being withdrawn its end must be tightly compressed lest its contents get into the larynx in passing.

(b) *Forced Feeding by the Mouth.*—This is generally carried out by the passage of a catheter into the stomach. The apparatus required is the same as that used for stomach washing, viz., a soft rubber catheter, connected, by a small piece of glass tube and a foot and a half of rubber tube, with a funnel large enough to hold from 2 to 6 oz. The most suitable size of catheter is a No. 12 or 14, and it should be provided with only one terminal opening.

The child is placed on his back, his head being held steady by an assistant. The left forefinger is then placed lightly on the tongue to depress it, while with the right hand the catheter is passed down the pharynx for 6 to 10 inches. The funnel is raised for a few minutes to allow the escape of gas, and the food is then poured into it and rapidly finds its way into the stomach. When the funnel empties the tube is tightly compressed and rapidly but gently withdrawn. If the withdrawal of the catheter is done slowly or clumsily it is apt to excite vomiting.

In infants who have no teeth, or only one or two, no gag is required. In older children some sort of a gag is necessary, as there is danger of the tube being bitten. In them the process is much more difficult and not so generally useful.

A simpler form of forced feeding, first recommended by Scott Battams,¹ is sometimes useful. For this all that is necessary is an ordinary glass syringe, to the nozzle of which 4 inches of rubber tube are attached. The child who is refusing food, or who for some reason is not to be allowed to suck, is laid on his back, the tube is passed towards the back of the mouth, and the liquid is gently injected. In older children, who clench their jaws, the tube may easily be passed backwards between the teeth and the cheek, and the liquid in this way readily reaches the pharynx.

Indications for Forced Feeding.—1. In the rearing of premature infants periodic feeding, either through the nose with a spoon or by means of a catheter passed through the mouth, has been found of great use (Tarnier).

2. Similarly, in young infants and others who are so weak that the effort of sucking and swallowing exhausts them, great benefit may be got from forced feeding, either through the nose or preferably by Mr Scott Battams' method.

3. In some cases of prostration (*e.g.*, in enteric fever) there is obstinate refusal of all food and medicine to an extent which seriously endangers life. These cases may be effectually treated by one of the methods of nasal feeding, or preferably by the syringe and short tube.

4. The same methods are sometimes serviceable in cases where swallowing is interfered with by pain due to ulceration of the mouth or throat.

5. Many years ago Kerley² drew attention to the fact that regular forced feeding by means of an œsophageal tube passed into the stomach was sometimes extremely useful in persistent vomiting in infants. Babies who are not able to retain a teaspoonful of fluid swallowed in the ordinary way can usually retain a much larger amount of nourishment if it is poured into the stomach through a catheter. The explanation of this is obscure, but of the occasional value of its application in practice there can be no doubt.

6. In cerebral cases, in cases of narcotic poisoning, and in convulsive conditions such as tetanus, where there is interference with the process of swallowing, life may be prolonged

¹ *Lancet*, 16th and 23rd June 1883, 1037 and 1084.

² "Gavage in the Treatment of Persistent Vomiting in Infants," *Archives of Pediatrics*, Feb. 1892, 85

and sometimes saved by forced feeding with a tube either through the nose or mouth. In diphtheritic paralysis affecting the pharynx, feeding through a tube is of the greatest possible value.

2. *Stomach-Washing, or Lavage.*

Methods and Apparatus.—A soft rubber urethral catheter or nasal catheter, size No. 12 to 14, connected with a funnel by 18 inches of tube, constitutes the best apparatus for stomach washing, as for gavage. Lukewarm $\frac{3}{4}$ -per cent. salt solution is probably the best fluid to use.

The patient is made to sit or lie on his mother's knee, with his face looking towards her left side and his clothes protected by a mackintosh sheet. A slight pressure on his chin generally makes him open his mouth, and the catheter is then passed gently backwards over the tongue, when swallowing movements commence and the tube is easily passed into the stomach. When the stomach is reached the funnel is momentarily raised to allow any gas present there to escape, and then lowered so that the stomach contents, if any, may be seen. Then the water is poured into it out of an ordinary jug.

In doing this one must be careful, especially in delicate children, not to over-distend the stomach by running in too much water at a time or by holding the funnel too high. When a sufficient amount of fluid has been introduced, at most 2 to 3 oz., the funnel is lowered, and the contents of the stomach rapidly fill it by syphon action and are emptied into a basin. The tube is then pinched to prevent the entrance of air, the funnel raised again and refilled, and the process of washing-out repeated. It should be continued until fragments of curd, etc., cease to be found in the returning fluid.

Indications of Stomach-Washing.—In many forms of dyspeptic derangement in infancy, stomach washing, with or without irrigation of the lower bowel, constitutes the most rational and successful preliminary treatment. Stomach lavage is specially valuable in pyloric stenosis and pyloric spasm.

It is also frequently, as we have seen, a valuable aid in the diagnosis and treatment of many of the diseases of the alimentary tract in infancy (pp. 327 *et seq.*).

The Mechanical Treatment of the Bowel.

Our objects in making local applications to the bowel are five in number. *Firstly*, to stimulate it to evacuate its contents. *Secondly*, to cleanse, soothe, or otherwise act on its mucous membrane. *Thirdly*, to soften retained fæces or to destroy parasites. *Fourthly*, to have food, fluid, or medicine absorbed from it without passing through the stomach; and *fifthly*, to reduce an intussusception or a prolapse of the rectum.

For these purposes we make use of suppositories, injections of various kinds, and irrigations.

Suppositories.—The simplest forms of local irritant that are used to stimulate the action of the bowel in constipation consist of small paper cones or conical pieces of soap. These are quite efficacious in many cases, and are unobjectionable provided the mucous membrane of the rectum remains quite healthy. Glycerine suppositories are more active; but, though they are useful in some cases, they must not be persevered with too long, as they are apt to give rise to catarrh of the rectum and to cause fissures and other troublesome complications.

Enemata.—Evacuant injections may be composed of plain soap and water, or thin gruel. During the injection the child should lie on his side, the fluid (at a temperature of 100° F.) should be allowed to run in very slowly and a certain amount of pressure exerted on the sides of the anus so as, if possible, to help its retention for a few minutes. In giving rectal injections to young children, a glass funnel and catheter are preferable to a Higginson's syringe.

Injections of glycerine are very efficacious in *constipation*. A teaspoonful may be used plain or mixed with an ounce of warm water. In obstinate cases, from 2 drm. to 1 oz. of castor oil, or from 1 to 4 drm. of turpentine, may be added to an ordinary soap-and-water injection.

Injections of laudanum and starch are very useful in relieving *tenesmus* and some forms of *diarrhæa*. From 2 to 3 minims of laudanum in a $\frac{1}{2}$ oz. of starch may be used for a strong child of a year old. In the insomnia of acute disease (e.g., in pneumonia), a rectal injection of warm water is sometimes very efficacious.

When *hard masses of fæces* accumulate in the bowel, a preliminary injection of olive oil ($\frac{1}{2}$ to 1 oz.) is useful in softening

them. It should, if possible, be retained for from four to six hours, and be followed by an ordinary soap-and-water enema.

The administration of *medicine* by the bowel is sometimes useful in an emergency when the patient is unable to swallow (*e.g.*, chloral in a case of convulsions).

Nutrient enemata consisting of 1 to 2 drm. glucose in $\frac{1}{2}$ to 1 oz. of water are of value when sufficient food by mouth is not retained or is contraindicated, *e.g.*, after operation for pyloric stenosis.

Saline rectal injections, given to allay thirst and to supply fluid to the tissues, form a most valuable therapeutic measure—especially in young babies and in severe dehydration.

The apparatus necessary for giving such injections consists in the barrel of a glass syringe attached to a small rubber catheter. The catheter is gently introduced for a couple of inches into the bowel, and the solution, which must be at blood heat, is allowed to run in very slowly. Normal saline solution (.9 per cent. NaCl), Ringer's solution (NaCl .7 per cent., KCl .01 per cent. and CaCl_2 .02 per cent.) or Hartmann's solution are all suitable. The catheter is compressed from time to time so as to moderate the flow. When the fluid has entered the bowel the catheter is withdrawn and the nates are held firmly together for a minute or two. When the rectum is very irritable, as often happens, it may be necessary for the nurse who is giving the injection to take half an hour or longer in its administration. In some very irritable cases the fluid will be ultimately well retained if the nurse allows only a few drops of the fluid to enter at a time and compresses the nates for many minutes at the end.

The right amount of saline to be injected varies in different cases, because it depends on the degree of sensitiveness of the particular child and not merely on the capacity of the rectum. Infants of a few weeks can usually retain from 6 drm. to 1 oz. and sometimes, with care, as much as 2 oz. Older children may retain much larger amounts. The injections may be repeated every four hours. Stimulants may be given in the injection, and are well borne if freely diluted.

Some of the indications for this method of treatment are as follows:—

1. In premature and extremely weak babies who are threatening to die from sheer debility, occasional rectal saline

injections or glucose nutrient enemata are a valuable aid to the use of the incubator and forced feeding.¹

2. They may also be useful in any diseased condition in newly-born or other weakly children (*e.g.*, birth injuries, septic conditions, hæmorrhage, etc.).

3. They are particularly valuable in cases where the child is unable to retain fluid taken by the mouth, as in pyloric hypertrophy and other conditions accompanied by severe vomiting, and after surgical operations when fluids are not allowed by the mouth.

4. Large enemata of plain water form a valuable diuretic measure in cases of acute nephritis.

Irrigation of the Lower Bowel.—To irrigate the colon, an ordinary douche apparatus, such as is used in obstetric practice, is required, with a large-sized rubber catheter at the end of the tube. The child is laid on a bed or table or on his nurse's knee in the lithotomy position, and a large mackintosh sheet is placed under him, draining into a pail below. The douche can be fixed 4 or 5 feet above the child. The catheter is then oiled and placed within the anus before the water is turned on. As the water flows the catheter is passed steadily upwards for 12 or 14 inches, if possible.

At six months old the colon will hold a pint without distension; and, at the age of two years, from two or three pints (Holt). As the irrigation proceeds the water begins to be forcibly expelled by the side of the catheter, and the process should be continued until the water which returns is tolerably clean. At least a gallon of water should be used for each irrigation. After the injection is over the water should be allowed to run out of the catheter, and it should be left in for a few minutes for this purpose. A considerable proportion of the injecting fluid is usually retained for some time.

Irrigation is useful in effecting the thorough clearing out of the lower bowel.

It is soothing to the mucous membrane and has a beneficial effect in various forms of diarrhœa, and also in some cases of recurrent colic. Plain water or saline solution seems to be as efficacious as an antiseptic solution.

Cases of catarrhal jaundice which are threatening to become

¹ Pery, "De la débilité, congénitale et acquise, des nouveau-nés," *Thèse de Bordeaux*, 1903.

chronic are sometimes greatly benefited by the daily administration of a cold water irrigation. For this purpose the water may be used between 65° and 85° F.

Intravenous Therapy.—In the child it is often essential that the therapeutic remedy be administered intravenously, as, *e.g.*, in the case of certain sera and the arsenical preparations for the cure of syphilis. In the sick and depleted infant, too, this is a most convenient route by which to supply fluid and nourishment, *e.g.*, saline and glucose solutions.

The veins of the neck, those at the bend of the elbow, the scalp veins or the longitudinal sinus may all be taken advantage of for this purpose, but they are not equally suitable at all ages. In the infant the scalp veins and longitudinal sinus will be found most convenient. In the syphilitic infant the scalp veins are as a rule unduly prominent and form a ready route, but in the depleted child these veins are little visible and the longitudinal sinus is the most satisfactory. The objections to the use of the longitudinal sinus are: (1) that if any drug escapes outside the lumen irritation of the brain results, and (2) that if sepsis is introduced a fatal meningitis ensues. Thrombosis of the sinus may also result. However, with care and an observance of strict aseptic precautions the operation is quite safe, but it should never be performed if there is a septic condition of the scalp. In the older child the bend of the elbow is the seat of election. The external jugular can be utilised at any age, but it has the disadvantage of being exceedingly mobile, which renders the operation of venipuncture difficult. The jugular vein, however, is specially valuable after the age of one year, when the scalp veins become less prominent and the fontanelle is closed, and before the age of three or four years when those at the bend of the elbow commence to be apparent.

One important point in carrying out this method of therapy is that the drug or solution should be prepared and all apparatus be in readiness before commencing. If a small amount of fluid only is to be introduced an ordinary 5 or 10 c.c. Record syringe and needle, No. 1, about an inch long and with the point sharpened at an angle of 45°, are all that is necessary. If, however, the amount of fluid is greater, *e.g.*, 100 or 200 c.c., or more as in the case of saline or glucose, then a small Luer-Kaufmann syringe with extra side-nozzle is a great advantage. In addition, a funnel to contain the fluid, with a piece of rubber tube attached

to connect up with the side-nozzle of the Luer-Kaufmann syringe, is necessary. A Murphy drip in the circuit is of great help in regulating the flow of the fluid. Some workers prefer an Ehrlenmeyer flask instead of a simple funnel. This flask is supplied with a two-holed rubber stopper supporting two pieces of glass tubing—one short, just reaching beyond the cork, and the other long, extending to the bottom of the flask. The tubing for conveying the fluid to the syringe is attached to the shorter glass tube, while the longer tube when the flask is inverted reaches above the level of the fluid and thus acts as an air-way and facilitates the flow. Temple Grey¹ recommends a double-walled Dewar's flask, which acts like a vacuum flask and retains the temperature of the fluid to be injected.

The serum or solution of the drug to be given is prepared and taken up into the syringe, or the solution (normal saline, Ringer's solution, Hartmann's combined solution, or 5 to 10 per cent. solution of glucose), sterile and at the proper temperature, is placed in the funnel or flask and held in readiness.

When it is decided to use the scalp veins the child is wrapped tightly in a towel or blanket to control the movements of the limbs, is laid on a high couch with the head resting on a firm and preferably wedge-shaped pillow, and held steadily in position by an assistant who grasps the occipital and frontal regions between the fingers and thumb of each hand, and at the same time applies slight pressure by his chest on the child's body. In this way the child is kept absolutely at rest, which considerably facilitates the operation. The most prominent and straightest veins are in the temporal region. The hair may require to be cut to improve the visibility, after which the part is thoroughly cleansed with ether. The assistant applies pressure with one thumb on the proximal portion of the vein just above the ear so as to distend it and render it more prominent. The needle is then inserted. One knows when the vein has been properly entered by the free flow of blood. When satisfied that the needle is properly within the lumen of the vein the syringe containing the solution of the drug is attached, the assistant releases the pressure on the vein and the solution is slowly injected.

If large amounts of fluid are to be introduced the Luer-Kaufmann syringe with Murphy drip and funnel or flask are

¹ F. Temple Grey, *Lancet*, 1932, ii., 127.

used. The whole length of tubing and apparatus is filled with the fluid so as to remove all danger of injecting air and the tube then clipped. The syringe with needle attached is introduced into the vein, suction applied and the syringe filled beyond the level of the side-nozzle, when, if the clip is released, the fluid can be allowed to flow under gravity by raising the funnel or flask above the level of the head.

For venepuncture of the longitudinal sinus the child is held as described above, except that the sides of the head are grasped by the assistant, with the occiput resting on the pillow. The same apparatus is used. The scalp in the region of the anterior fontanelle is prepared as before and the sinus entered in the middle line at the posterior angle of the fontanelle with the point of the needle directed towards the occipital prominence. Special needles with an extra large head or flange to enable a better grasp, and so a steadier hold, are supplied, but they are not necessary. The rate of flow of the fluid can be modified by raising or lowering the level of the funnel and by varying the pressure of the clip on the supply tube.

In the case of the external jugular vein, pressure applied in the supraclavicular fossa causes its engorgement and brings it into greater prominence. When the median basilic vein at the bend of the elbow is selected, it is wise to apply a light tourniquet and get the patient to open and shut the hand several times, or the arm may be passively flexed and extended at the elbow. These manœuvres often bring into prominence a vein which at first cannot be seen, and they will at least make it palpable even when it is not visible.

Blood Transfusion.—Before using any individual's (*the donor*) blood for the purpose of transfusion it must be tested (*matched*) against that of the patient (*recipient*) to see if they will mix without hæmolysis of one or the other blood resulting. It is generally stated, however, that in the case of the new-born this precaution is not necessary.

Matching of the bloods is carried out by observing the effect of the recipient's serum on the red-blood corpuscles of the donor, and also, although this is less necessary, the effect of the donor's serum on the recipient's corpuscles. A saline suspension of red corpuscles of each blood is obtained by allowing a few drops of blood to mix with a few cubic centimetres of normal saline. Serum can be obtained either from blood got by venepuncture

and centrifugalised, or by puncture of the finger and collected in a capillary tube as is done for carrying out the Widal reaction. To perform the test a drop of the serum of one individual and of the saline suspension of corpuscles of the other individual are mixed on a glass slide and observed under the microscope for fifteen minutes. If no clumping of the corpuscles occurs within this time the bloods are said to match and to be safe to mix. Through the agency of the British Red Cross Society, and in connection with many hospitals, a list of suitable donors is now available.

When the suitable donor is found the transfusion may be done directly from arm to arm ; but this method is now discarded for that of using citrated blood. For the use of citrated blood special apparatus is necessary and this should be prepared, assembled and in readiness before commencing the operation. One or more 100 c.c. all-glass syringes may be employed not only to collect the blood but to introduce it into the vein of the patient. Before use 10 c.c. of a sterile 3·0 per cent. solution of citrate of soda are drawn into each syringe so as to counteract clotting. When the syringe is filled it is gently rotated so as to thoroughly mix the blood and the citrate and the whole kept warm until required by wrapping it in a moist warm towel.

A more convenient receiver for the blood is an Ehrlenmeyer flask. This is fitted with a rubber stopper pierced by two holes, through which two pieces of glass tubing are inserted. One of the pieces of glass tubing reaches almost to the bottom of the flask and the other only a short distance beyond the cork. By a piece of rubber tubing and adapter the longer glass tube is connected with the needle for venepuncture, and to the short tube there is attached another length of rubber tubing interrupted by a glass bulb containing cotton-wool or other filter material and supplied with a mouthpiece. This latter tube is useful for applying suction and thus creating a negative pressure to facilitate the flow of blood. Instead of an Ehrlenmeyer flask Temple Grey recommends a double-walled Dewar's flask, which has the advantage that it acts like a vacuum flask and retains the temperature of the mixture. Before allowing the blood to enter the flask all tubing is washed through with a 3 per cent. solution of citrate of soda, and 10 c.c. for every 3 oz. of blood to be collected are introduced into the flask. When the flask is to be used for discharging the blood, a fresh cork, also with two

holes and two different-sized pieces of glass tubing as before, is inserted. This time the tubing with the mouthpiece is attached to the longer tube. The short tube is attached by means of rubber tubing which is interrupted by a Murphy drip to the side-nozzle of a Luer-Kaufmann syringe, the whole being previously washed through with the citrate solution and all air removed by filling completely with citrated solution or saline and the tube then clipped. When the flask is inverted the longer tube reaches above the level of the blood and permits of the entrance of air or the creation of a positive pressure to facilitate the flow. The vein selected (external jugular, longitudinal sinus, or median basilic at the bend of the elbow according to the age of the patient) is entered by the needle attached to the Luer syringe and suction applied until blood fills the syringe beyond the level of the side-nozzle, when the plunger is fixed, the clip on the supply tube released, and by the action of gravity blood allowed to flow slowly from the inverted flask through the Murphy drip at the rate of 60 to 300 drops per minute according to the age of the patient. 60 drops per minute represents a flow of 60 c.c. or 2 oz. in twenty minutes, which is a safe rate for infants. 300 drops per minute means that half a pint is discharged in twenty minutes, which is a suitable rate for the adult. For older children a rate between these two extremes may be chosen.

If the blood has been collected in a large syringe (100 c.c.) this is attached by rubber tubing to the side-nozzle of the Luer-Kaufmann syringe, which is filled with the patient's blood beyond the level of the side-piece and then the donor's blood slowly injected.

APPENDIX A

METHOD OF CASE-TAKING

Name—Age—Parent's occupation—Address—By whom recommended.
Date of admission. Date of examination. Date of discharge.

Complaint, on account of which the child has been brought. Its duration.

Family History.—Health of parents and near relatives (rheumatism, tubercle, nervous and mental disease, syphilis, etc.)—Mother's health during pregnancy, and facts as to previous pregnancies—Miscarriages—Number of other children alive, their ages and health; number dead, their ages and cause of death.

Hygienic Surroundings.—Dwelling-house (situation, size, ventilation, light, warmth, dampness)—Amount of open-air exercise.

Previous Health.—Nature of labour—Condition and weight of child at birth—Feeding during infancy (breast or bottle—exact details)—Later feeding—Dates of teething and of beginning to walk and speak—Usual state of digestion and bowels—Sleep—Signs of congenital syphilis—Fits (number and date of occurrence)—Rickets—Attacks of diarrhœa, vomiting, bronchitis, sore throat, otorrhœa, enlarged glands—Infectious diseases and age at which they occurred (measles, whooping-cough, scarlet fever, chicken-pox, etc.).

Present Illness.—Date of commencement, and whether sudden or gradual—Health immediately before—Supposed or possible causes (injury, chill, improper feeding, etc.)—Symptoms noticed in order of appearance, *e.g.*, languor, wasting, irritability, debility, loss of appetite, thirst, vomiting, diarrhœa or constipation, cough (its character and time of occurrence), pain, laryngismus, convulsions (general or local), perspiration, fever, breathlessness, sore throat, disturbed sleep, drowsiness, etc.

State on Examination.—Height—Weight.

A. General Inspection and Palpation.

Appearance (if healthy or otherwise)—Nutrition and development—Complexion (anæmia, cyanosis, jaundice, etc.)—State of skin (dryness, moisture, eruptions, desquamation, pigmentation, œdema)—Attitude, expression, demeanour, temper.

Shape of head, and state of its ossification (fontanelle, cranio-tabes)—Facial irritability—Hair—Eyes, nose, and ears (formation of, and if any discharge from)—Neck—Shape of thorax, abdomen, back, and limbs (especially the hands)—Enlarged glands—Evidence of rickets, syphilis, and tuberculosis.

Character of voice, cry, and cough—Rate and character of respiration (if noisy, dyspnœic, or painful)—Movements of alæ nasi—Rate and character of pulse—Temperature.

Palpation of abdomen (tenderness, resistance, fluid, size of liver and spleen, tumours, etc.).

B. Further Detailed Examination of Systems. N.B.—The systems found to be affected should be taken first, but *in all cases* the urine, heart, lungs, abdomen, throat and ears must be examined and their condition recorded.

Respiratory System.

Thorax (form, measurement, movements, retraction).

Palpation (position of heart's apex-beat, fremitus).

Auscultation—Percussion—Exploratory puncture—Sputa.

Circulatory System.

Inspection of præcordia, epigastrium, and neck (form, pulsation, etc.).

Palpation (position, character, and force of heart's apex-beat and other pulsations, thrills). Auscultation—Percussion.

Hæmopoietic System.

Spleen (palpation, percussion), lymphatic glands, thymus, thyroid, blood.

Genito-Urinary System.

Micturition (control of bladder, dysuria)—Examination of kidneys, bladder, and external genitals (phimosis, hernia, etc.).

Urine (amount and characters). Microscopic examination.

Skin.

Texture and Elasticity. Eruptions (distribution, type, itchiness, enlargement of neighbouring lymphatic glands).

Locomotor System.

Joints, epiphyses, shafts, muscles.

Nervous System.

Cranium (size and shape)—Fontanelle (shape, edges, tension, level, pulsation, and size). Spine (form, pain, rigidity, retraction of head). Mental condition—drowsiness, stupor, coma, disturbances of sleep, irritability, excitement, delirium, attention, memory, intelligence, speech.

Motor Functions—Muscles (development of), paralysis.

Involuntary movements (tremor, chorea, spasm—tonic, or clonic, convulsions).

Voluntary movements (strength and co-ordination).

Holding up head, sitting up, standing, walking.

Electrical reactions.

Reflexes, superficial, deep, organic.

Sensory Functions—subjective sensations, sensibility to touch, pain, and temperature, tenderness (local or general).

Vasomotor and Trophic Functions.

Eyes—sight, photophobia, conjunctiva, cornea, pupils (size, shape, and reaction), nystagmus, strabismus, ptosis, muscular paralysis, ophthalmoscopic examination.

Ears—pain, discharge, hearing, otoscopic examination.

Nose—Smell, taste.

Digestive System.

Lips, mouth, tongue, gums, teeth, palate, tonsils, fauces, pharynx, adenoids.

Thirst, appetite, vomiting, state of bowels, vomited matter, fæces.

Abdomen (further inspection, palpation, percussion). Rectal examination.

DIAGNOSIS. TREATMENT AND PROGRESS. RESULT.

IN CASE OF DEATH, COPY OF PATHOLOGIST'S REPORT.

APPENDIX B

PERIODS OF INCUBATION AND INFECTIVENESS OF INFECTIOUS DISEASES

The following facts are taken partly from the Clinical Society's *Report on the Periods of Incubation and Contagiousness of certain Infectious Diseases*, London, 1892, and from the *Code of Rules* issued by the Medical Officers of Schools Association, eighth edition, London, 1923.

Diphtheria.

Incubation Period.—This generally lasts two to five days, and never exceeds ten days. Quarantine, twelve days.

Infective Period.—The patient may be infectious for nearly four weeks. He may return to school in four weeks, provided that convalescence is completed, that there is no longer any sore throat, nor any abnormal discharge from the throat, nose, ears, or eyes, no cutaneous pustulation and no albuminuria, and that at least three successive bacteriological examinations of both the pharyngeal and nasal mucus for the specific bacillus, made at intervals of not less than two days, have given negative results—each examination having been made not less than three hours after the discontinuance of local antiseptic applications.

Persistence of Infection.—The infection can be retained in clothes, carpets, and other fomites for months, perhaps for years.

Influenza.

Incubation Period.—This usually lasts three or four days, but varies from a few hours to five days in duration.

Infective Period.—The patient may convey infection during the whole course of the illness, *i.e.*, for a week or ten days.

Measles.

Incubation Period.—This usually lasts for ten days, rarely only for four or five, or for as long as fourteen days. Generally the rash appears on the fourteenth day from exposure to infection. Quarantine, sixteen days.

Infective Period.—Measles is very infectious during the invasion, and probably remains so during the whole of the acute attack. The infection may last for almost two weeks from the beginning of the rash.

Persistence of Infection.—Fomites are probably capable of retaining infection for a very short time only.

Mumps.

Incubation Period.—The interval between the exposure to infection and the onset of parotitis is generally three weeks, or a day or two more or less. It is occasionally as long as twenty-three days, or, more rarely, as short as fourteen days. The beginning of the prodromal stage is so difficult to ascertain, and so uncertain in duration, that the beginning of the illness is usually dated from the onset of the parotitis. Quarantine, twenty-four days.

Infective Period.—This may begin with the beginning of the prodromal stage, which may last as much as four days; it is very active during the onset of the parotitis; it diminishes progressively from that time, and may be regarded as having ceased any time after a fortnight, provided a clear week has elapsed since the subsidence of all swelling.

Rubella.

Incubation Period.—This lasts usually for eighteen days, but may be as long as twenty-one, or rarely as short as nine days. Quarantine, twenty days.

Infective Period.—The patient may be infectious for two or three days before the rash appears, as well as when it is out. The infection probably passes off within five days of the appearance of the rash, provided there is no persistence of nasal or other symptoms.

Scarlet Fever.

Incubation Period.—This usually lasts for more than twenty-four, and less than seventy-two hours. It is occasionally less than a day, and sometimes longer than three days; it never exceeds seven days. Quarantine, ten days.

Infective Period.—It is infectious from the onset of the earliest symptoms; but probably ceases to be so long before desquamation is completed, unless there is any nasal discharge, otorrhœa, suppurating wound, or

eczematous patch. When any of these is present, the discharge from them may continue very infectious for a long time after desquamation has ceased. When none of these sources of discharge is present, the patient is probably free from infection by the end of the fourth week.

Persistence of Infection.—This infection is readily preserved in, and conveyed by, fomites.

Varicella.

Incubation Period.—This lasts generally for fourteen days, but may be a day less, or four or five days more. Quarantine, twenty days.

Infective Period.—The patient is infectious at least as soon as the rash appears. A convalescent patient may convey the infection to others, so long as any scabs are left on the body or scalp. The infection may be conveyed in clothes, but is certainly not long retained by them.

Whooping-Cough.

Incubation Period.—This lasts usually for fourteen days, but may extend from seven to nineteen days. Quarantine, twenty-one days.

Infective Period.—Whooping-cough is very infectious during the invasion, and while the spasmodic cough is developing. By the end of the fifth week of the cough, the infection has probably always ceased.

Persistence of Infection.—Fomites are probably capable of retaining infection for a very short time only.

APPENDIX C

DIRECTIONS TO MOTHERS RESPECTING PARALYSED CHILDREN

In Use at the Great Ormond Street Children's Hospital.

Clothing.

The paralysed parts must be kept warm day and night.

Knitted woollen stockings to come up above the knees must be worn.

If these do not keep the limbs warm, woollen overalls must be worn outside the stockings.

The overalls must be lined, if necessary, with cotton wadding quilted to them.

For the night a flannel sack made the shape of the leg, coming up to the top of the thigh and lined with cotton wadding, is best.

Rubbing.

This must be done for a quarter of an hour twice daily.

Lay the child on a bed.

1st.—Rub the paralysed leg from the foot right up to the top of the thigh. Rub upwards only. Put the broad part of your hand on

the back of the child's leg. In rubbing the thigh, put your hand, first on the back of the thigh and afterwards on the front, but always rub upwards, and be sure to go as high as the child's loins. Whilst rubbing with your right hand, hold the child's foot with your left. Use for rubbing any kind of oil.

2nd.—Take hold of the child's leg with your two hands just above the ankle. Rub round the leg with your two hands in opposite directions, as though you were wringing out sheets. Work up the leg and thigh, from the foot up to the top of the thigh, in the above manner.

3rd.—Take the child's calf with your two hands. Put your fingers to the back of the leg and your thumbs to the front. Squeeze the soft parts out between your fingers and thumbs, so as to flatten the leg out and make it as wide as possible. Work right up the leg and thigh in this manner.

4th.—Put your right hand over the front of the child's knee, and your left hand under the foot. Push up the foot, and holding your right hand in front of the child's knee you will prevent yourself doing any harm. You want, if possible, by pushing the foot, to make the child push against your left hand with all his might. *This is the most important of all the exercises.*

5th.—Flip every part of the leg and thigh with your fingers, so as to make the whole of the limb quite red and warm.

6th.—Gently rub up and down all over. This will take away the stinging which was left by the last movement.

Baths.

Once a day let a large jugful of hot water, containing two handfuls of salt, be poured down the leg and thigh.

Then pour about half the quantity of cold water over the leg and thigh.

Then, rub thoroughly with a towel until the limb is perfectly warm and dry.

APPENDIX D

DIRECTIONS TO THE MOTHERS OF YOUNG CHILDREN WHO ARE MENTALLY DEFECTIVE¹

Your child needs to be carefully taught to do things that other children do without teaching. In time he may learn to do them quite well if you only persevere.

Remember that improvement *cannot* be sudden; it can only come gradually by getting him to do over and over again little things that he is not good at. Notice, therefore, what things he cannot do as well as other children, and try to teach him to do them better one by one. Do not go

¹ This leaflet may be obtained from the Central Association for Mental Welfare, 24 Buckingham Palace Road, London, S.W. 1; price 4d. a dozen.

on doing for him anything that you can possibly get him to do for himself—such as feeding or dressing.

Encourage him especially in doing those things that he finds a *little* difficult, but do not give him anything to do that is quite too hard for him. Utter failure will discourage him, while success in *anything* that is not mischief will do him a great deal of good.

Always encourage anything harmless that he does of his own accord. Such things please him far more than what you tell him to do, and are also better for him ; but *never* let him even begin to get into a habit of making faces, or of making any noises that you would not like your other children to learn.

If he seems to notice too little, encourage him to look at, and listen to, or handle anything that he is taken up with. Any sort of interest helps to brighten him.

Do your best to keep his body as strong as possible by carefully seeing to his food and clothing, and by taking him into the fresh air as much as you can.

Nobody knows how much he may improve ; that will depend largely on the amount of trouble and patience you spend on him.

APPENDIX E

FORMULÆ

Balnea.

FORM. 1.

Hot 98°-106° F.

FORM. 2.

Tepid 85°-92° F.

FORM. 3.

Cold 65°, reduced to 45° F. or lower

Balneum Alkalinum.

FORM. 4.

Sodium carbonate $\frac{1}{4}$ oz.

To each gallon of water at 90° F.

Balneum Sinapis.

FORM. 5.

Mustard $\frac{1}{2}$ oz.

To each gallon of water at 98° to 106° F.

(The mustard to be enclosed in a bag and squeezed.)

Balneum Sulphureum.

FORM. 6.

Sulphurated potash $\frac{1}{4}$ oz.

To each gallon of water at 98° F.

(To be used in other than a metal bath.)

Alopecia.

FORM. 7.

Acid. lactic. . . . $\bar{3}$ i to $\bar{3}$ i

Olei ricini $\bar{3}$ ii

Spt. vin. rect. . . . $\bar{3}$ iv

To be applied, with caution, once daily.

FORM. 8.

Sol. Amm. fort. . . . $\bar{3}$ ss

Chloroformi $\bar{3}$ ss

Olei sesam. . . . $\bar{3}$ ss

Olei limonis $\bar{3}$ ss

Spt. rosmarini ad. $\bar{3}$ iv

To be used cautiously until tolerance is acquired.

Anaemia.

FORM. 9.

Ferri et ammon. cit.	. . .	grs. ii
Pot. cit.	. . .	grs. ii
Liq. arsenicalis	. . .	min. ss
Aq. menth. pip.	. . .	ad. ℥i

FORM. 10.

Ferri et ammon. cit.	. . .	grs. iiss
Pot. cit.	. . .	grs. ii
Aq. menth. pip.	. . .	ad. ℥i

FORM. 11.

Vin. ferri. cit.	. . .	℥ss to ℥i
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FORM. 12.

Ferri et ammon. cit.	. . .	grs. ii
Tinct. nuc. vom.	. . .	min. i
Syrupi aurantii	. . .	min. x
Aq.	. . .	ad. ℥i

Anorexia.

FORM. 13.

Ichthyol	. . .	grs. ii
Syrupi	. . .	min. v
Glycerini	. . .	min. v
Aq.	. . .	ad. ℥i

Thrice daily.

Bronchitis.

FORM. 14.

Amm. carb.	. . .	gr. ss
Pot. bicarb.	. . .	grs. ii
Tinct. ipecac.	. . .	min. iiss
Syr. tolu	. . .	min. x
Aq. carui	. . .	ad. ℥i

FORM. 15.

Tinct. opii camph.	. . .	min. iiss
Acid. hydroch. dil.	. . .	min. i
Vin. ipecac.	. . .	min. iiss
Glycerini	. . .	min. x
Aq. carui	. . .	ad. ℥i

FORM. 16.

Tinct. opii camph.
 Oxymel scillae
 Syrup. tolu āā. min. xx
 (Not to be used for children under
 12 years.)

Bronchitis and Asthma.

FORM. 17.

Pot. iodid.	. . .	gr. i
Tinct. bellad.	. . .	min. iiss
Tinct. lobeliae (ether).	. . .	min. ivss
Aq. chlorof.	. . .	ad. ℥i

Carminative.

FORM. 18.

Sod. bicarb.	. . .	grs. ii
Spt. ammon. arom.	. . .	min. ii
Glycerini	. . .	min. x
Aq. carui	. . .	ad. ℥i

Chronic Intestinal Catarrh.

FORM. 19.

Sod. bicarb.	. . .	grs. iii
Tinct. nuc. vom.	. . .	min. i
Inf. gent. co.	. . .	ad. ℥i
(½ hour to 10 minutes before each meal.)		

FORM. 20.

Pot. iodid.	. . .	gr. i
Sod. bicarb.	. . .	grs. x
Tinct. catechu.	. . .	min. v
Inf. senag.	. . .	ad. ℥i

Colic and Flatulence.

FORM. 21.

Sod. bicarb.	. . .	grs. iii
Spt. amm. arom.	. . .	min. i
Spt. chlorof.	. . .	min. i
Syrupi	. . .	min. x
Aq.	. . .	ad. ℥i

FORM. 22.

Sod. bicarb.	. . .	grs. ii
Papain (Finkler)	. . .	gr. i

Before each meal.

FORM. 23.

Chloral. hyd.	. . .	grs. iiss
Aq. laurocerasi	. . .	min. x
Syr. prun. virgin.	. . .	min. x
Aq.	. . .	ad. ℥i

Every three hours.

Constipation.**FORM. 24.**

Magnesii sulphat.	.	.	grs. iiss
Acid. sulph. dil.	.	.	min. i
Ferri sulphat.	.	.	gr. ss
Syrupi zingib.	.	.	min. ii
Aq. menth. pip.	.	.	ad. $\bar{3}i$

Thrice daily after food.

FORM. 25.

Sod. bicarb.	.	.	$\bar{3}ii$
Syrupi rhei	.	.	$\bar{3}ii$
Ext. cascara sagrada liq.	.	.	ad. $\bar{3}iv$

$\bar{3}i$ after each meal.

FORM. 26.

Tinct. bellad.	.	.	min. v
Tinct. nuc. vom.	.	.	min. ss
Syrupi sennae	.	.	min. x
Inf. gent. co.	.	.	ad. $\bar{3}i$

Thrice daily.

FORM. 27.

Aloin	.	.	gr. $\frac{1}{4}$
Strychnine	.	.	gr. $\frac{1}{60}$
Ext. bellad.	.	.	gr. $\frac{1}{8}$

FORM. 28.

Ext. cascara sagrada liq.	.	.	min. v
Mucil. tragacanth.	.	.	q. s.
Syrupi zingib.	.	.	min. iv
Aq. anethi	.	.	ad. $\bar{3}iv$

Dentifrice.**FORM. 29.**

Thymol	.	.	gr. ss
Olei gaulther.	.	.	min. iss
Olei rosmarii	.	.	min. iss
Glycerini	.	.	$\bar{3}v$
Spt. rect.	.	.	min. v
Liq. rosae dulc.	.	.	$\bar{3}v$
Aq.	.	.	$\bar{3}v$
Pulv. cret. praecip.	.	.	$\bar{3}i$

Make into a paste.

Febrifuge Mixture.**FORM. 30.**

Sod. sal.	.	.	grs. iss
Quin. sulph.	.	.	gr. $\frac{1}{4}$
Glycerine	.	.	min. x

Spt. chlorof.	.	.	$\bar{3}ss$
Mucil. tragacanth.	.	.	q. s.
Aq.	.	.	ad. $\bar{3}i$

Liniments.**FORM. 31.**

Olei teribinth.	.	.	$\bar{3}i$
Lin. camph. co.	.	.	ad $\bar{3}ii$

FORM. 32.

Olei amber.	.	.	$\bar{3}ss$
Olei caryoph.	.	.	$\bar{3}ss$
Olei olivae	.	.	ad $\bar{3}ii$

Mouth-Wash.**FORM. 33.**

Pot. tart. (acid)	.	.	grs. ii
Sod. chlorid.	.	.	grs. ii
Sacch. alb.	.	.	gr. $\frac{1}{4}$
Aq.	.	.	ad. $\bar{3}i$

One ounce to be used to wash out the mouth after each meal, and no water used after. (GIBBS).

FORM. 34.

Pot. tartrat. acid.	.	.	grs. ii
Acid. tartar.	.	.	gr. i
Sacch. alb.	.	.	gr. $\frac{1}{4}$
Olei limonis	.	.	min. iii
Aq.	.	.	ad. $\bar{3}i$

(PICKERILL).

Nasal Obstruction.**FORM. 35.**

Sod. bicarb.	.	.	grs. x
Acidi boric.	.	.	grs. x
Sod. chlorid.	.	.	grs. v
Aq.	.	.	ad $\bar{3}i$

Ringworm.**FORM. 36.**

Sulph. precip.	.	.	$\bar{3}ss$
Hyd. amm. chlor.	.	.	$\bar{3}ss$
Acidi Salicyl.	.	.	grs. xx
Lanolin	.	.	$\bar{3}ss$
Vasilin.	.	.	$\bar{3}ss$

To be thoroughly massaged into the affected spots for at least ten minutes twice daily.

FORM. 37.

Hyd. oleat.	grs. xl
Acid Salicyl.	grs. x
Lanolin	℥ss
Vasilin.	℥ss

As in Form. 36.

Stomatitis (ulcerative).

FORM. 38.

Pot. chlor.	grs. ii
Syr. aurant.	min. xv
Aq.	ad. ℥i

Three or four times daily.

FORM. 39.

Liq. arsenicalis.	℥ii
Tinct. ipecac.	℥ii
Glycerini	℥ii
Aq. menth. pip.	ad. ℥ii.
Dilute min. xv. with water, and apply as paint to mucous membrane of mouth every six hours.	

Thrush.

FORM. 40.

Sod. biborat.	pt. i
Glycerine	pts. vi

To be used as a paint.

Tonics—Bitter.*Acid.*

FORM. 41.

Acid. nit. dil.	min. iss
Glycerini	min. x
Inf. calumbae	ad. ℥i

FORM. 42.

Acid. nit. dil.	min. i
Decoct. cinchonae	ad. ℥i

Alkaline.

FORM. 43.

Sod. bicarb.	grs. ii
Tinct. calumb.	min. iv
Sph. chlorof.	min. ss
Inf. amantii	ad. ℥i

FORM. 44.

Tinct. cinchonae co.	min. x
Glycerini	min. v
Aq.	ad. ℥i

Vermifuge.

FORM. 45.

Santonini	gr. i
Zingib. pulv.	gr. i
Jalap	grs. iii
Sulphur	grs. iv
Confectii sennae	ad. ℥i

(Dose for child 2 to 5 years.)

Whooping-Cough.

FORM. 46.

Antipyrine	grs. ii
Amm. bromid.	grs. v
Tinct. bellad.	min. v
Aq. menth. pip.	ad. ℥i

FORM. 47.

Ext. thymi vulgaris liq.	min. xv
Aq.	ad. ℥i

Lotions.*Alkaline boric lotion.*

FORM. 48.

Sod. bicarb.
Sod. chlorid.
Sod. biborat. āā.

℥i to ℥ii to be added to a pint of
warm water.*Calamine and Carbolic Acid.*

FORM. 49.

Acid. carbol.	℥ss
Calamin.	℥i
Zinc. oxid.	℥ii
Glycerini	℥iii
Aq. calcis	℥iv
Aq.	ad ℥iv

Boracic acid lotion.

FORM. 50.

Sod. biborat.	grs. viii
Aq.	ad. ℥i

Calamine lotion.

FORM. 51.

Calamin.	grs. xl
Zinc oxid.	grs. xx
Acid. boric.	grs. xv
Glycerini	min. xx
Aq.	ad. ℥i

Lotions---*continued.**Coal-tar lotion.*

FORM. 52.

Liq. picis carb. . . . min. xii
 Aq. ad. ʒi

Lead lotion.

FORM. 53.

Sol. plumbi. subacet. fort. . min. vi
 Aq. ad. ʒi

Lead and opium lotion.

FORM. 54.

Tinct. opii min. xxx
 Lotio. plumbi ad. ʒi

Red lotion (Hey's Wash).

FORM. 55.

Zinc. sulph. grs. ii
 Tinct. lavend. co. . . . min. x

Ointments (antiseptic).*Furunculosis.*

FORM. 56.

Sulph. prepar. grs. xv
 Acid. carbol. min. xv
 Olei amygdale min. iii
 Paraff. moll. alb. . . . ʒi

Impetigo.

FORM. 57.

Hydrarg. ammon. . . . grs. v
 Paraff. moll. alb. . . . ʒi

Cracked Nipples.

FORM. 58.

Lanolin 4 pts.
 Paraffin moll. 3 pts.
 Tinct. benzoin. co. . . . 1 pt.

Scabies.

FORM. 59.

Sulphuris ʒi
 Balsami peruv. ʒss
 Ung. simplicis ʒi

FORM. 60.

β-Naphthol pts. xv
 Cret. prep. pts. x
 Saponis moll. pts. l
 Adipis. pts. c

Seborrhæic dermatitis.

FORM. 61.

Ung. Bismuth. Oleat. B.P.
 (For face and head, to be spread
 on mask made of lint.)

Poultices.*Cataplasma Acidi Borici et Amyli.*

FORM. 62.

Mix ʒi powdered boric acid with ʒss starch and make into thick cream with water. Add to this, while constantly stirring, a pint of boiling water. If necessary, the mixture is again brought to the boiling-point to ensure complete gelatinisation.

When cold the jelly is spread thickly on sheets of soft cotton material, covered with thin muslin, and applied to desired surface, being changed every few hours.

Cataplasma Lini.

FORM. 63.

Add gradually to 10 oz. of boiling water, with constant stirring, 4 oz. of crushed linseed.

Cataplasma Lini cum Sinapis.

FORM. 64.

Mustard, 1 part ; crushed linseed, 5 parts ; boiling water, a sufficient quantity. Mix the mustard into a thick cream with lukewarm water ; proceed by mixing linseed separately as for a plain linseed poultice ; finally stir the two mixtures well together and spread on linen cloth.

APPENDIX F

RECIPES

Arrowroot Water.—Rub up a teaspoonful of arrowroot with a tablespoonful of cold water until smooth; pour on it, while stirring, a pint of boiling water, and boil for five minutes.

Barley Jelly.—Put two tablespoonfuls of *washed* pearl barley into a pint and a half of water, and slowly boil down to a pint; next, strain out the barley, and let the liquid settle into a jelly. Two teaspoonfuls of this, dissolved in 8 oz. of warmed and sweetened milk, are enough for a single feeding.

Barley Water.—Put two teaspoonfuls of *washed* pearl barley into a pint of cold water, boil down to two-thirds of a pint, and strain through muslin. It should be made twice a day, as it may turn sour with keeping.

Beef Tea.—Mince one pound of lean beef, and add to it a pint of cold water and 10 drops of dilute hydrochloric acid; let it stand for two or three hours, with occasional stirring, and then simmer from ten to twenty minutes.

Brandy and Egg Mixture.—Rub up the yolks of two eggs with a tablespoonful of white sugar, and add four tablespoonfuls of brandy and eight of cinnamon water. Dose, a teaspoonful to a tablespoonful.

Bread Jelly.—Take a thick slice of bread (4 oz.) two or three days old, so as to be dry and sweet, and of seconds flour (since this is richer in protein and phosphates than the finest white flour); place it in a basin of cold water and allow it to soak for six or eight hours. It is then to be taken out, and all the water squeezed out of it. The pulp is then placed in a pint of fresh water and gently boiled for an hour and a half. The thick gruel thus made is strained, rubbed through a fine hair sieve, and allowed to grow cold, when it forms a fine, smooth, jelly-like mass. It should be freshly prepared, night and morning, for it will not keep long. Enough of the jelly is mixed with warm water, previously boiled, to make a food of the consistence of thin cream (about one full tablespoonful to 8 oz. of water) so as to pass readily through the bottle; a little white sugar may be added (W. B. Cheadle, *Artificial Feeding of Infants*, 3rd edition, 1894, 84).

Ketogenic Diet.—The ketogenic diet is one in which there is an inadequate proportion of carbohydrate (anti-ketogenic element) to permit of complete combustion of the fat (the ketogenic element). One-half of the protein is burned as carbohydrate so that it is equally ketogenic and anti-ketogenic in action. In the normal adult diet (fat 100 grammes, protein 100 grammes, and carbohydrate 500 grammes) the proportion of the ketogenic to the anti-ketogenic elements is 1 to 3.7. Ketosis or incomplete combustion of fat does not usually occur until the ratio is 1 to 1 and in the child till this is more than 2 to 1.¹ In the two illustrative diets on facing page, supplying roughly 1000 calories and suitable for most children if confined to bed, the proportion of the ketogenic to the anti-ketogenic elements is 3 to 1 and 5 to 1.

There is, as previously mentioned (p. 554), no danger in connection with such a diet. Symptoms of acidosis never develop and within a few days the child accommodates himself to the altered proportion of ketogenic and

¹ M. J. Brown and G. Graham, *Arch. Dis. Child.*, 1926, i., 357.

DIET A.—*Ratio of ketogenic to anti-ketogenic elements 3 : 1.*

	Fat grms.	Carbo- hydrate grms.	Protein grms.	Calories.	Ketogenic units.	Anti- ketogenic units.
Breakfast—						
Egg 1.	6	...	6	80	9	3
Milk, 200 c.c. . . .	6	9	6	114	9	12
Diabetic roll
Butter, 15 grms. . .	12	108	12	...
11 a.m.—						
Milk 200c.c. with 25 c.c. } 20 per cent. cream . . }	6 5	9 ...	6 ...	114 45	9 5	12 ...
Dinner—						
Bacon, 50 grms. . .	32	...	5	310	34	3
Cabbage or } Tomato, 100 grms. }	...	5	...	20	...	5
Bovril q.s.
Tea—						
Tea with 25 c.c. 20 per cent. cream . . .	5	45	5	...
Diabetic roll
Butter, 30 grms. . .	25	225	25	...
Total . . .				1061	108	35

DIET B.—*Ratio of ketogenic to anti-ketogenic elements 5 : 1.*

	Fat grms.	Carbo- hydrate grms.	Protein grms.	Calories.	Ketogenic units.	Anti- ketogenic units.
Breakfast—						
Tea with 25 c.c. 20 per cent. cream . . .	5	45	5	...
Bacon, 50 grms. . .	32	...	5	310	34	3
Diabetic roll
Butter, 15 grms. . .	12	108	12	...
11 a.m.—						
Cream (20 per cent.) 100 c.c.	5	45	5	...
Dinner—						
Fish, 60 grms. . .	0.18	...	10.3	43	5	5
Cabbage or } Tomato, 50 grms. }	...	2.5	...	10	...	2.5
Milk, 200 c.c. . . .	6	9	6	114	9	9
Tea—						
Tea with 25 c.c. 20 per cent. cream . . .	5	20	5	...
Diabetic roll
Butter, 30 grms. . .	24	216	24	...
Cheese, 10 grms. . .	3.6	0.4	2.7	46	5	1.7
Total . . .				957	104	21

anti-ketogenic elements and the ketosis rapidly diminishes and ultimately disappears. The only difficulty is that the amount of fat makes the diet nauseating and many children either refuse to take it or are sick.

Malt Infusion.—Mix three full tablespoonfuls of crushed malt thoroughly with half a pint of cold water in a jug. Allow the mixture to stand overnight—ten or twelve hours. Decant off the supernatant liquid carefully from the sediment, and strain it through two or three folds of muslin, until it comes through fairly clear and bright. This should make about 6 oz. of malt infusion. It should be kept in a well-corked bottle, and a few drops of chloroform are to be added to preserve it. A dessertspoonful may be added to the food before it is taken, or be sipped along with it (Sir Wm. Roberts, *Collected Contributions on Digestion and Diet*, 1891, 224).

Oatmeal Water.—Take one tablespoonful of coarsely ground oatmeal, add a pint of water, and simmer gently for an hour; strain, and add sufficient water to replace that which has evaporated.

Raw Meat-Juice.—Mince finely the best rump steak; add cold water in the proportion of one part of water to four of meat. Stir well together, and allow to soak for half an hour, cold. Forcibly express the juice through muslin by twisting it.

Rules for a Salt-free Diet.—(1) No salt is to be used in cooking, nor is any to be eaten at meal times. (2) Ordinary bread, including cakes and scones from the baker, fish (except fresh-water fish), salt butter, cheese, all forms of dried, smoked, salted, and otherwise preserved food, such as tinned meat, bacon, dried fish and the like, and sauces and pickles, must be avoided. (3) Not more than a pint of milk must be taken in the day. (4) Soups made from ordinary meat stock contain large quantities of salt; without this they are unpalatable. The soup for a patient undergoing a strict course of treatment should be made from vegetables, with a milk stock. (5) Apart from these restrictions, the patient may eat what he likes. He may have from $\frac{1}{4}$ to $\frac{1}{2}$ lb. of meat, or its equivalent as poultry or fresh-water fish, per day, and vegetables, cereals, bread made without salt, one or two eggs, etc., as his appetite demands. Tea and coffee are not prohibited. The liberal diet which is thus possible is much less irksome than the "light diet" to which many nephritis patients are restricted, and children quickly become habituated to the absence of salt. After a time, the diet may be relaxed somewhat, the addition of ordinary bread being the most grateful change (J. S. Fowler in *Garrod, Batten, and Thursfield's Diseases of Children*, 1913, 637).

Sherry Whey.—Ten oz. of milk are heated until just boiling, then $2\frac{1}{2}$ oz. of *cooking* sherry are added and heat applied again until the mixture begins actually to "boil up," when it is removed from the fire and allowed to stand three minutes. The curd is then strained off through a twofold layer of butter muslin.

Veal Tea.—Take a pound of minced veal, free from fat; mix with a pint and a half of water or barley water; heat in a slow oven for three hours; strain and skim.

White of Egg Water.—Take the white of a fresh egg; cut it in various directions with a clean pair of scissors. Shake it gently in a bottle, with a pinch of salt and a half pint of cold water. Strain through muslin.

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